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OBSERVATIONS ON VENOUS PRESSURES AND THEIR RELATIONSHIP TO CAPILLARY PRESSURES.

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Reprinted from the Journal of Physiology.
Vol. XVI. Nos. 3 *&* 4, 1894.

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[*From the Journal of Physiology. Vol. XVI. Nos. 3 & 4, 1894.*]

OBSERVATIONS ON VENOUS PRESSURES AND THEIR RELATIONSHIP TO CAPILLARY PRESSURES.

By W. M. BAYLISS, B.A., B.Sc. and ERNEST H. STARLING, M.D., M.R.C.P. (Plates VIII. IX. X. and 3 Figs. in Text.)

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The experimental results which we wish to bring forward are largely such as might be predicted by anyone with a knowledge of the elementary principles of the circulation. Our justification in bringing them forward however is that they have not been so predicted, and it was only after obtaining the results that we asked ourselves why they had not occurred to us before. In fact they seem to form part of a forgotten or disregarded chapter in the physiology of the circulation, although they are of great importance for the question of pressure in the capillaries of the abdominal organs and therefore for the physiological processes of secretion and transudation which take place in these organs.

Thus even in Heidenhain's experiments on the relationship of lymph formation to blood pressure, the arterial blood pressure is the only factor measured and is spoken of as if it were synonymous with, or varied directly as, the capillary pressure. Now this assumption, although true in certain particular cases, would only be true generally if the vascular system were similar to a series of tubes through which water is flowing from an inexhaustible reservoir. Here changes in the height of the head of pressure would cause corresponding changes in the pressure at any part of the system. The vascular system however is a closed system of tubes with definite capacity and containing a definite amount of fluid, and the pressure in any given capillary area cannot be at once estimated from a tracing taken from the femoral or carotid arteries.

In the first place, between capillaries and the arterial pressure is situated the peripheral resistance, i.e. the arterioles, so that, unless we are certain that these arterioles do not alter in size, we cannot say that a rise of general pressure will occasion any corresponding rise in the capillaries beyond the peripheral resistance. If we have to be content with a single determination of pressure, it would be better to take the pressure in the vein coming from the set of capillaries, the condition of which we are investigating. Here there is in most cases no unknown or varying resistance situated between the capillaries and the point at which we are investigating the pressure, so that the venous pressure would give us a better clue to the variations of the capillary pressure than the observations on arterial pressure. As a matter of fact however, neither of these methods suffice in themselves. Since we cannot conveniently measure capillary pressure directly and do not know the rate of fall of pressure in the arterioles, the only method which will give us reliable information as to the variations of capillary pressure, is to take the pressure on both sides of these capillaries, that is to say, to take arterial and venous pressures simultaneously. Even under these circumstances however there is a number of variable factors which must be taken into consideration before we can decide as to the existence or direction of a change in the intracapillary pressure.

It will be simpler in the first place to take the case in which the distribution of resistance in the area under consideration undergoes no change, i.e. the arterioles neither contract nor dilate. In this case, if both arterial and venous pressures rise together, or if one pressure rises while the other remains constant, we may say with certainty that the capillary pressure is increased. In the same way if both pressures fall, or if one falls while the other remains constant, the capillary pressure must be diminished. If on the other hand, the pressures vary in different directions, if, for instance, the venous pressure goes up while the arterial pressure sinks, it must be entirely a matter of surmise as to what occurs in the capillaries, and the pressure here may be rising, sinking, or remaining constant. If the arterial pressure rises while the venous sinks, we can in one case assert that the capillary pressure is diminished. This is the case in which the rise in arterial and the fall in venous pressure is due to constriction of the arterioles in the part under observation. In other cases in which there is a change in the calibre of the smaller arteries, the determination of a change in the capillary pressure is a matter of considerable difficulty. We will take for example the splanchnic area. In the diagram *a* to *b* repre-

sents the fall of pressure in arteries, *b* to *c* from arterioles to capillaries, and *c* to *d* from capillaries to trunk of portal vein. We will assume in

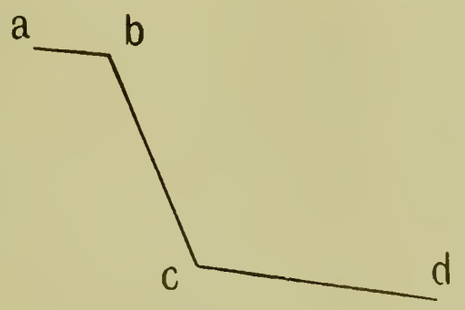


Fig. 1.

the first place that the resistance from *c* to *d* does not vary. The fall of pressure from *c* to *d* will be then directly proportional to the velocity of the blood from *c* to *d*. On stimulation of the splanchnic nerves there is a constriction of the arterioles and therefore a greater fall of pressure from *b* to *c*, and a less flow of blood through the system. The pressures both at *c* and *d* will be diminished. In consequence of the constriction however the general arterial pressure is raised. This rise is not sufficient to drive the normal amount of blood through the resistance and therefore $p(c)$ and $p(d)$ will be still below normal. Now if we put in sufficient resistance at *d* the rate of fall of pressure will be altered and we may by this means increase both $p(c)$ and $p(d)$ not only to normal but above normal value. In the body such a resistance is interpolated on stimulation of the splanchnics, since a constriction of the hepatic branches of the portal vein is thereby produced. We observe in fact on stimulation of the splanchnics, a rise of pressure in the portal vein as well as in the systemic arteries. Will this increased resistance in the liver raise the capillary pressure to normal or above normal? The factors on which the answer to this question depends will be made clearer if we take numerical values for the various pressures.

$p(d)$ normally is equal to 7 mm. Hg. If we assume that the resistance from *c* to *d* is equal to that presented by the liver capillaries, we may take $p(c)$ as 13 mm. Hg. This fall of pressure is proportional to the velocity of the flow from *c* to *d*.

On stimulation of the splanchnics, v. Basch has shown that the flow of blood from the portal vein is diminished to $\frac{1}{3}$ of what it was before. The fall of pressure from *c* to *d* is therefore also diminished to one-third of what it was before, and is reduced from 6 to 2 mm. Hg. If in consequence of the increased resistance in the liver, the venous pressure

were raised to normal, i.e. if $p\,(d)$ during splanchnic stimulation equals 7 mm. Hg, then $p\,(c)$ during the same time would be 9 mm. Hg—so that we should have a fall of capillary pressure, although the arterial pressure is raised and the venous remains constant. As a rule however it is found that the portal vein pressure is raised above normal, and may rise to 12 mm. Hg. Under these circumstances $p\,(c)$ would be 14 mm. Hg, and would be practically unchanged. So that we may have a rise of pressure on both arterial and venous sides without any corresponding rise of capillary pressure. And if we had taken the capillary pressure at 20 mm. Hg instead of 13 mm., there would be an actual fall of capillary pressure during splanchnic stimulation in spite of the simultaneous rise in arteries and veins. In fact in this case there are too many unknown factors to be certain of the presence or direction of any change in capillary pressure.

Exactly the same difficulty is met with in the converse case, namely a fall of one or both pressures associated with a dilatation of the arterioles. Here as in the former case the determining factor will be the change in the velocity of the blood current produced by the altered circumstances. We may have a fall of capillary pressure, or the diminution of resistance may more than counterbalance the fall of pressures, so that the capillary pressure will be increased. We consider a special case of this description later on (Effects of section of the cord).

A fall of either or both pressures associated with arterial constriction will of course diminish capillary pressure; and the converse case, a rise of arterial or venous pressure or of both, associated with arterial dilatation, will cause an increase in the capillary pressure.

In our experiments we have estimated the pressure in the femoral artery, in the inferior cava, and in the portal vein. In our earlier experiments, about 20 in number, we measured the pressure in one of these veins together with the femoral arterial pressure. In our later experiments, of which we give the protocols at the end of this paper, all three pressures were recorded simultaneously. In this way we hoped to throw some light on the variations in the capillary pressure produced in the abdominal organs (intestines &c.) and in the liver, by various procedures, such as obstruction of the larger blood vessels, stimulation of the vagi or splanchnics, asphyxia and so on.

Method of experiment.

In all experiments, medium-sized dogs were used, varying in weight from about 7 to 11 kilos. These received half an hour before the

experiment an injection of 1 to 2 grains of morphia. During the experiment they were kept completely anaesthetic with small amounts of A. C. E. mixture. In no case was curare given.

Preparation of Splanchnics. These were exposed from the back, by incisions half an inch below and parallel to the last ribs, and were ligatured and divided directly after their passage through the crura of the diaphragm. When it was desired to stimulate them they were put on Ludwig electrodes, and in any case the wounds were then stitched up, leaving only the ends of the electrodes showing. In most cases, on exposing the splanchnics, one to three smaller splanchnics on each side may be seen passing towards the suprarenal plexus. Sometimes the upper one of these runs so close to the large splanchnic that it may be ligatured and put on the electrode with this nerve. Where this was not possible, all the smaller splanchnics were divided or torn through. In Exp. 1 the smaller splanchnics could not be seen, so that we had to be content with the division of the large splanchnics.

This method of exposing the splanchnics is fully described by Cyon[1]. He makes the limitation however that the procedure is only applicable to small dogs. This limitation does not exist. The operation, though rather more tedious, is possible on dogs weighing as much as 20 kilos. In large and fat animals however it is necessary to make a larger opening, since the wound is much deeper than in small dogs.

When the splanchnics were to be excited, both sets of electrodes were connected in circuit with the secondary coil of the inductorium (Mayer's, with Ewald's contact breaker).

The dog was then turned over, and the rest of the preparation proceeded with. The vagi were prepared in the neck in the usual manner. The femoral artery and common femoral vein were then dissected out on the left side, and furnished with François Franck cannulae in their central ends. In this way we measured the arterial pressure in the external iliac artery, and the venous pressure in the beginning of the inferior cava. Care must be taken to insert the venous cannula above the entry of the deep femoral vein, since there is often at this point a valve which prevents reflux from the iliac vein. It will be observed that in Exp. 13, neglect of this precaution prevented any observation of the femoral venous pressure, and the experiment had to be interrupted for the reinsertion of the cannula.

Arrangements were then made for the obstruction of the thoracic aorta, inferior vena cava, or the portal vein.

[1] *Physiologische Methodik*, p. 193.

For the first two vessels obturators consisting of German silver cannulae were used. These were closed and rounded off at one end, and were pierced near this end with small holes. Over this end a finger cap of india-rubber was tied. The other end of the cannula, which was open, had a rubber tube attached to it for the insertion of the nozzle of a small syringe. The extent to which the rubber capsule might be distended was tried before the experiment, and the same quantity of water which was found necessary was injected during the experiment.

In order to obstruct the inferior vena cava, the obturator was introduced through the right external jugular vein. For the obstruction of the aorta Heidenhain introduced the obturator through the right carotid artery. In our small dogs however we found this difficult, and killed two by sending the end of the obturator into the pericardium through the wall of the aorta. We then thought of introducing the obturator through the external iliac artery, and found this extremely easy. In all cases therefore in which the aorta was to be obstructed, the external iliac artery on the right side was exposed by an incision just above Poupart's ligament, and the obturator introduced in this way. The peritoneum was not opened.

The portal vein was exposed by a small incision in the middle line of the abdomen, and a ligature passed round it and attached to a ligature staff, so that the vein could be occluded or released at pleasure.

The last step in the operation was that for taking the pressure in the portal vein. For this purpose the splenic vein was used. A small incision was made on the left side of the rectus abdominis muscle, beginning close under the ribs. Two fingers were then introduced and the spleen drawn out. All the arteries going to the spleen were then ligatured[1], and a cannula (François Franck) inserted in the largest of its veins. The intestines were not exposed at all during this operation[2]. The cannulae were then connected with the manometers. The arterial pressure was registered on a Hürthle's kymograph (with slow movement) by means of a mercurial manometer. The manometer tube had a diameter of 2·5 mm.

The venous cannulae were connected by rubber tubes to two manometers made of barometer tubing with an internal diameter of

[1] If this is not done, the spleen swells enormously, and may give rise to troublesome bleeding if accidentally pricked or scratched.

[2] A similar method for the determination of the portal pressure was adopted by v. Basch. *Ludwig's Arbeiten*, 1876.

1·5 mm. These were fastened on to wooden stands and had scales divided into millimetres behind them. The cannulae and manometers were filled from the side tube of the cannulae with 25% $MgSO_4$ solution which was coloured blue with a little methylene blue. The height of the fluid in the manometers was adjusted so as to be about equal to the expected venous pressure. A solution of 25% $MgSO_4$ was also used to fill the arterial cannula. By using this solution with narrow bore manometers the danger of clotting was entirely obviated. In no case in experiments extending over 2 or 3 hours did clotting ever occur in the venous cannulae. Below the tracing of arterial blood pressure a time marker connected with a clock registered 10, 15, or 30 second intervals. In the time marking circuit was also placed an electric bell, which sounded every time that the signal made a mark on the tracing.

The experiment being ready, the manometers being connected and the drum started, one of us sat down opposite the venous manometers, and read off their height every time the bell sounded, while the other wrote down the figures. Letters were now and then put against the venous reading and scratched on the arterial reading in order to be certain that the venous and arterial readings were synchronous.

At the end of the experiment, the venous and arterial abscissae were taken and recorded, and later on the arterial pressure was carefully measured and written out in parallel columns with the two venous pressures. Some of these records we give at the end of this paper. *The venous pressures are given in millimetres of* $MgSO_4$ *solution, and the arterial pressure in millimetres of mercury.* The solution of $MgSO_4$ had a specific gravity of 1046, so that it was almost exactly 13 times lighter than mercury. Hence the venous pressures must be divided by 13 to reduce them to mm. Hg.

Theoretical Considerations.

The text which is illustrated and confirmed by the whole of our observations is furnished by Weber in his classical paper "Ueb. die Anwendung der Wellenlehre auf die Lehre vom Kreislaufe des Blutes[1] &c." Speaking of his diagram of the circulation, which with the addition of a funnel in the venous part, is similar to that which we give in fig. 2, he says:—"We see on the simplified model of the circulation that the pump (the heart) cannot increase the mean

[1] Reprinted in *Ostwald's Klassiker der exacten Wissenschaften*, p. 30 (from *Berichte ü. d. Verhandl. der königl. Sächs. Gesellschaft der Wiss. z. Leipzig*, 1850).

pressure exerted on the walls of the system of tubes by the fluid contained within them. It can in fact only give rise to an unequal distribution of the pressure, by diminishing the pressure in the veins by pumping fluid out of them and increasing the pressure in the arteries to a corresponding extent by pumping the fluid into them. The mean pressure of the fluid in this model can only be increased by distending the tubes to a larger extent by the injection of more fluid into them."

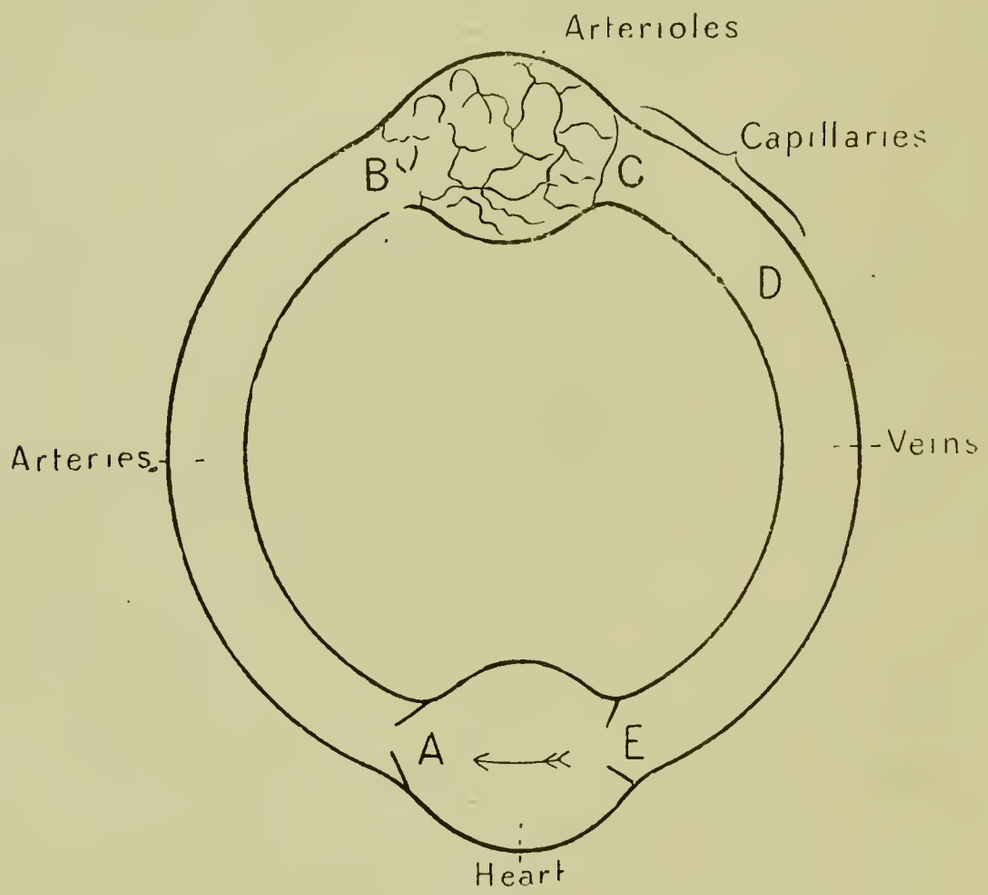


FIG. 2.

Thus we may take Fig. 2 to represent the vascular system, which has a definite capacity, and contains a definite quantity of blood. If the heart AE is not acting and the fluid is motionless, the pressures at all parts of the system will be the same. In dogs under these circumstances the pressure will be from 5 to 10 mm. Hg. We may take this as the mean general blood pressure in Weber's sense. If now the heart begins to act, it pumps blood from the veins into the arteries, so that the latter become distended at the expense of the former. In this way the arterial pressure rises above and the venous pressure sinks below the mean pressure of the system, and in a system of this description the higher the arterial, the lower will be the venous pressure, whether the rise in arterial pressure be conditioned by an

increase in the heart's activity or by an increase in the peripheral resistance. The capillaries being on the venous side of the peripheral resistance, the pressure in them will move with that of the veins, so that the higher the arterial, the lower will be the venous, and therefore the capillary pressure. (This latter statement is only partly true, and presupposes that all the resistance from *C* to *E* is negligible compared with that from *B* to *C*. It is only true for an area such as the liver, and our generalisation is only partially true for other areas. We have taken the extreme case however in order to show the fallacy of supposing that capillary pressure must in all cases vary directly with arterial pressure.)

Changes in heart beat or in peripheral resistance, as Weber points out, will not increase or diminish the "mean pressure" but will only cause a variation in the distribution of the pressure.

In the body however there is another factor which we have not taken into account. A change in the peripheral resistance brought about by a contraction of the arterioles is never alone. The contraction of these vessels not only increases the peripheral resistance but also diminishes the total capacity of the system, so that we have the same effect produced as if more fluid were poured into a system of the original capacity. Thus in the body an increase in peripheral resistance must always connote a rise in the "mean pressure" of the system brought about by the diminution of its capacity. We are no longer able to say that increased arterial pressure is necessarily accompanied by diminished pressure on the venous side. The amount of blood forced out of the arterioles (in which we must include the portal vein and its branches [1]) may be sufficient to distend both arterial and venous trunks more than before, so that we get a simultaneous rise of pressure in both arteries and veins.

The rise of arterial pressure occasioned by vaso-motor activity has thus to be explained by the coincidence of two factors:

(*a*) Increased peripheral resistance,

(*b*) Diminished capacity of blood vessels:

and this has been the ruling idea in most of the papers on the subject emanating from the Leipzig laboratory [2]; although in this country the second factor has not perhaps had sufficient importance ascribed to it.

[1] Mall. "Der Einfluss des Systems der Vena Portae auf die Vertheilung des Blutes." *Du Bois' Archiv*, 1892, p. 409.

[2] Cp. Slavjansky. *Ludwig's Arbeiten*, 1873, p. 665; v. Basch. *Idem*, 1875, p. 373; Mall. *loc. cit.*

Section I. Some effects of the vascular nerves on the circulation.

Section of the cord.

The effect of this procedure is seen by comparing Experiments 10 and 11. At the close of Exp. 10, the pressures were

Fem. Art.	Portal Vein.	Fem. Vein (Inf. Cava).
125 mm. Hg [1].	97 mm. $MgSO_4$ sol.[1]	60 mm. $MgSO_4$ sol.[1]

The cord, which had been previously exposed, was then divided just above 1st dorsal vertebra.

The pressures after 10 mins. were then found to be (Exp. 11):

Fem. Art.	Portal Vein.	Fem. Vein.
62 mm.	73 mm.	61 mm.

We thus see that the effect of the diminished peripheral resistance, which would tend to raise the venous pressure, is exactly counterbalanced by the increased capacity of the system caused by the vaso-motor paralysis. Hence the pressure in the vena cava (peripheral end) is unaltered, while everywhere else there is a fall of pressure. The "mean pressure" of the system is diminished by section of the cord. (In many cases, as in the experiment given in the note at the end of this paper, there is also a distinct fall in the vena cava pressure.)

Effect on capillary pressure.

There is a simultaneous fall of pressure in aorta and portal vein. This however is accompanied and occasioned by relaxation of the arterioles all over the body, including the splanchnic area. As we have shown above, if this diminution of resistance is so great that the velocity of the blood through the capillaries is increased it is impossible to say with certainty whether the capillary pressure will be diminished, increased, or unaltered. It is necessary then to know how the amount of blood flowing through the splanchnic area is affected by section of the cord.

We have been unable to find any experiment bearing on this question, so have performed two experiments on the subject.

A cannula was put in the splenic vein, and a ligature on a staff

[1] These of course will be understood in the subsequent quotations of pressure.

round the portal vein. At a given signal the clip on the splenic vein was taken off and the portal ligature pulled tight. The blood flowing from the splenic vein was collected in graduated cylinders, the amount obtained in 5 secs. being measured.

Then the spinal cord was divided and the outflowing blood measured again.

Exp. Dog about 12 kilos.
Amount in 5 secs. 4—5 (pause of 2 mins.) 3·5—6—9.
Cord then divided opposite 7th cervical vertebra. Pause of 5 mins.
Amount in 5 secs. 2—3·8—4—4·5—4·4 (pause of 3 mins.).
„ „ „ 2—2—2—2·2—4—4—**5·5**—**4·5**—**4·4**—2·4—2·8—
2—**7** (in 40 secs.) 2—1·2—1·2—**1·2**—2.

(The darker figures signify that the cord was excited while these amounts of blood were being obtained.)

We see from this experiment, that after section of the cord the amount of blood flowing through the splanchnic vessels in a given time is less than before. We have therefore lowering of arterial and venous pressures with diminished velocity of blood flow, and therefore the pressure in the capillaries of the intestines &c. must be also diminished. The pressure in the hepatic capillaries will also be slightly diminished, since there is a diminution of portal pressure, while the inferior cava pressure is unaltered or slightly diminished.

Effect of cardiac inhibition in animals with divided cord.

When a vagus is excited and the heart thereby brought to a standstill, the effect on the circulation is rather complex, and of the changes occurring in the pressures at various points, some are not conditioned *directly* by the mere cessation of the circulation. Hence it will be convenient to deal first with the simple case of vagus inhibition in which the vessels are removed from the action of the vaso-motor centre by section of the cord in the lower cervical region.

The effects of complete standstill of the heart produced by vagus stimulation after section of the cord are given in Exp. 11, and are shown graphically in Diagram I. (Plate VIII.). In this experiment the pressures before excitation of the vagus were

Fem. Art.	Portal Vein.	Fem. Vein.
58 mm.	73 mm.	62 mm.

On exciting the peripheral end of the right vagus the heart stopped dead. Ten seconds later the pressures were

Fem. Art.	Portal Vein.	Fem. Vein.
20 mm. Hg.	72 mm.	68 mm. $MgSO_4$ solution.

The arterial pressure then gradually sank, while the venous pressures rose slightly so that at the end of three minutes after the heart standstill had begun the pressures were

Fem. Art.	Portal Vein.	Fem. Vein.
$6\frac{1}{2}$ mm. (= 84 mm. $MgSO_4$)	83 mm.	82 mm.

so that complete equilibrium was established between the pressures at all points of the vascular system. As is not unfrequently the case when the vagus is stimulated after division of the cord, the heart did not recommence beating spontaneously, and it was necessary to introduce the fingers into the abdomen and pinch the heart rhythmically in order to restore the circulation. When the heart was once more beating regularly the pressures were

Fem. Art.	Portal Vein.	Fem. Vein.
42 mm.	81 mm.	46 mm.

It is worth noting in this experiment that the venous pressures were almost unaltered by the stimulation of the vagus, the portal pressure only rising 1 cm. and the femoral venous pressure only 2 centimetres of water.

In this dog therefore $6\frac{1}{2}$ mm. Hg represents the mean pressure of the system, that is, the pressure exerted by the contained blood on the walls of all the blood vessels when circulation is at a complete standstill.

With regard to the effect of the vagal stimulation on capillary pressures, it is evident that the slight rise in portal pressure is negligible compared with the large fall (66 cm. water) in the arterial pressure, so that there must be a considerable fall in the pressure in the intestinal capillaries.

In the hepatic capillaries the pressure will be unchanged, or rather will be very slightly raised, since there is a small rise of pressure both in the portal vein and the inferior cava.

Excitation of the splanchnic nerves.

The effects of strong excitation of the splanchnics are given in Exp. 5, and the three curves are plotted out in Diagram II. In

this experiment both splanchnics were excited simultaneously by a strong induction current during 130 seconds. The alteration caused in the arterial pressure is the typical one, as described by Johansson[1]. It first rises quickly from 94 mm. to 116 mm., then falls slightly to 111 mm., then rises gradually to 132, where it remains constant until the end of the excitation. It begins to fall at once on discontinuing the excitation, and in the course of one minute reaches its former level. The portal pressure, after a very slight fall, which was too transitory to be recorded within the 10 seconds, begins to rise, gradually at first, and then more quickly to 170 mm., falling then to 160 mm., where it remains constant until the end of excitation. At this point the portal and arterial pressures move in opposite directions, the portal rising slowly, until in 40 seconds after the end of excitation, it has attained the height of 190 mm. It then falls gradually, reaching its original level in about two minutes. The behaviour of the portal pressure on splanchnic excitation is a constant phenomenon and has occurred in all our experiments. Exactly the same thing was found by von Basch in his work on the action of the splanchnic nerve on the circulation. At that time, the existence of vaso-motor nerves to the portal vein was unknown and von Basch attempts, not very successfully, to account for the variations in the portal vein pressure by variations in the calibre of the arteries supplying blood to the portal area. Lately Mall[2] has shown that stimulation of the splanchnics causes active contraction of the portal vein and its branches, and ascribes a great part of the rise in arterial pressure which occurs on stimulation of the splanchnics to the squeezing of blood out of the portal vein. It is evident that the rise of portal pressure is conditioned by the contraction of its branches in the liver. This constriction must outlast the constriction of the arterioles, hence we have a moderate rise of pressure during the stimulation of the nerve, when very little blood is flowing through the arteries into the vein, and a much larger rise of pressure on cessation of the excitation when the relaxing arterioles allow a full flow of blood into the still constricted portal vein. Von Basch[3] made another series of observations on the outflow of blood from the opened splenic vein during the various stages of the excitation, and found that during excitation the flow of blood was diminished to a third of what it had been before excitation, so that the venous pressure is raised during a period when the blood flowing into it is diminished.

The pressure in the vena cava is slightly increased from 68 mm. to

[1] *Du Bois' Archiv*, 1891, p. 103. [2] *loc. cit.* [3] *loc. cit.*

84 mm. and remains at this height during the whole of excitation, falling again to 68 mm. at the end of excitation. This behaviour of the vena cava pressure is only a special instance of the possible event which we mentioned earlier. The diminution in capacity of the whole vascular system is more than enough to counteract the effects of the increased resistance. As we pointed out above, mere increased resistance would lower venous pressure. The effect of the diminution in capacity has however been to raise the "mean pressure" of the system, so that there is increased distension both of arteries and veins, with a corresponding rise of pressures.

In Exp. 5, one of the vagi was intact and the heart was slowed as usually occurs when the splanchnics are stimulated. One might be inclined to ascribe the rise of venous pressure to this slowing of the heart, were it not that in other experiments where both vagi were divided, we still obtained a slight rise on stimulation of the splanchnics.

Effect on capillary pressures.

Intestinal capillaries. The effect of stimulation of the splanchnics on the pressure in the intestinal capillaries has been discussed above, and we have shown that we have not yet sufficient data to decide what change, if any, takes place. At the end of stimulation however we have relaxed arterioles with a normal arterial pressure and increased portal pressure, so that there must be, for a short time after cessation of the stimulation, an increased pressure in the intestinal capillaries.

Hepatic capillaries. The pressure in the portal vein is increased, while that in the vena cava is practically constant. Hence we might say that the pressure in the capillaries must be increased. But in this case the rise in portal pressure is entirely due to constriction of the branches of the portal vein in the liver, so that if the vena cava pressure was lowered there would be a lowered capillary pressure. The vena cava pressure however being constant or slightly increased, we cannot assume any appreciable fall of pressure in the hepatic capillaries, and it is probable that the pressure in these capillaries is very little affected by stimulation of the splanchnic nerves.

Effects of Asphyxia.

We give protocols of three experiments in which the animals were killed by asphyxia (Exp. 4, 6, and 12). In Experiment 4 the dog was normal, with the exception that both vagi had been divided.

We will first describe this experiment, and shall then discuss the causation of the various events in the pressure curves, which are plotted out in Diagram III.

The course of the arterial pressure is too well known to need description. In this experiment it was high to commence with (120 mm. Hg). Asphyxia was induced by opening both pleural cavities. Forty seconds after the first pleural cavity was opened the pressure began to rise and went up to 140 mm. The rise of pressure lasted exactly one minute. The mercury then began to sink, rapidly at first and more slowly later on, till death occurred from cardiac failure.

The portal pressure rose from 84 mm. $MgSO_4$ solution to 124 during the rise of arterial pressure. There is then a slight fall to 114 mm. and then as the arterial pressure falls, the portal pressure rises steadily to 144 mm. This height is attained two minutes after the arterial pressure has begun to fall. Then the portal pressure begins to fall, but very gradually, so that at the end of the experiment it is still 92 mm. The pressure in the inferior vena cava runs a somewhat similar course, the first rise of pressure being however not so marked as the corresponding rise in the portal vein. At the commencement of the experiment it is 36 mm. During the arterial rise, it also rises to 70 mm., and then as the arterial pressure begins to fall, the pressure in the vena cava rises enormously until it equals that in the portal vein. It then begins to fall gradually with the portal pressure, the two pressures being approximately equal. At the end of the experiment the vena cava pressure was 92 mm.

How are we to explain these results? It is evident that we have here two factors at work. The first is the constriction of the arterioles (chiefly splanchnic) and portal vein in consequence of the asphyxial stimulation of the vaso-motor centre, and the second is the gradual failure of the heart causing cessation of the circulation and the equalisation of pressures in all parts of the vascular system.

These two factors have long been known to effect the well known arterial changes in asphyxia, but their coaction is also necessary for the production of the increased venous pressure. The curves may be interpreted as follows.

The first event is a stimulation of the vaso-motor centre, acting especially on the splanchnic area, and causing constriction of arterioles and portal vein, and a general rise of arterial and venous pressures. Then, as the heart begins to fail and the circulation to be slowed, the pressures

in all parts of the circulation tend to approach more and more to the "mean pressure," that is the pressure exerted by the blood on all parts of the blood vessels when the blood is at rest. In consequence however of the still active vaso-motor centre, the total capacity of the system is diminished, and the amount of blood being constant it follows that the mean pressure must be increased above normal. The 144 mm. to which the venous pressures rise, represents or approaches then the mean pressure of the constricted system, and not the mean pressure of the vascular system in a normal condition. Hence the large venous rise that one gets in asphyxia is conditioned chiefly by the vaso-motor excitation. Without this super-added constriction of the blood vessels, without this lowering of the capacity of the system, asphyxia gives rise to only a trifling rise of venous pressure—a rise comparable to that which we studied as the result of exciting the vagus after section of the spinal cord. The final gradual fall of venous pressures has probably two causes. One is the gradually increasing distension of the heart's cavities, which would tend to empty the vascular system. The other, which is more problematical, is the gradual giving way of the contracted vessels, as the vaso-motor centre and nerves suffer in their turn from the continual asphyxia.

It will be noticed that under these conditions it takes longer to establish equilibrium between arterial and venous pressures than in the case where all the vessels are relaxed from section of the cord. Thus in this experiment, when the heart had long ceased beating and seven minutes after the arterial pressure had begun to fall, the pressure in the arteries was still two or three centimetres of water higher than in the veins[1].

The necessity for the coaction of the two factors above mentioned for the rise of venous pressure may be shown very simply. We may cut out the vaso-motor centre from the asphyxia experiment by section of the cord, or we may merely prevent its action on the abdominal vessels by section of both splanchnic nerves.

In either case we shall find that the venous rise produced by asphyxia is now comparatively insignificant.

The effect of asphyxia after division of the cord at the 1st dorsal vertebra is shown in Diagram IV., and details of the experiment are given in Protocol 12. Here there is no rise of arterial pressure. The portal pressure at the beginning of the asphyxia rose from 89 to 105, but this was probably caused by the increased movements of the diaphragm,

[1] Cp. de Jäger. This *Journal*, Vol. VII. p. 174, 1886.

since the pressure dropped again as soon as these movements became slower. It then fell slowly with the arterial pressure to 65 mm. The vena cava pressure merely rose very gradually from 48 to 64 as the heart failed.

We see here then that without vaso-motor stimulation, mere cessation of the heart's beat, whether conditioned by asphyxia or by vagal stimulation, is insufficient to cause more than a slight rise of pressure in the great veins.

In the same way the large rise of venous pressure may be abolished by section of the splanchnic nerves. An experiment of this nature is recorded in Protocol 6. In this experiment both vagi and splanchnics were cut. Asphyxia was then induced by opening both pleural cavities. The arterial pressure began to drop very soon, and this fall was accompanied at first by a slight fall of both venous pressures (? vaso-dilator excitation). We then endeavoured to imitate the natural course of the pressure curves by stimulating both splanchnics with strong induced currents. The first stimulation caused a rise of all three pressures.

Thus at the beginning of the experiment the normal pressures were

Fem. Art.	Portal Vein.	Fem. Vein.
54	96	65

Just before stimulation they had sunk to

Fem. Art.	Portal Vein.	Fem. Vein.
25	83	68

After exciting the splanchnics for one minute the pressures were

Fem. Art.	Portal Vein.	Fem. Vein.
26	110	98

On discontinuing the excitation all three pressures sank and after another 80 seconds were

Fem. Art.	Portal Vein.	Fem. Vein.
10	84	84

On excitation we got a very slight rise in the portal and femoral vein pressures to 95 and 90 respectively.

These results, although by no means equalling those obtained when the vaso-constriction is accomplished in the normal way by means of the centre, nevertheless serve to illustrate the action of the splanchnics

on the venous pressures and bear out what we have said above as to the action of these nerves.

Action of the vagus.

We are now in a position to examine the pressure curves obtained when the peripheral end of the vagus is excited in an animal otherwise normal. We give protocols of two experiments of this nature (Exps. 3 and 10).

In Exp. 3 the right vagus was intact, while the left vagus was divided, and the peripheral end put on Ludwig's electrodes. The excitation lasted exactly two minutes. The heart stopped dead, directly the current was sent into the nerve, and did not beat for 80 seconds. It then began to beat very slowly, and this slow pulse lasted till the exciting current was stopped.

During the stoppage of the heart the arterial pressure sank from 90 mm. Hg to 26, 20, 16, and then 10 mm. Hg, and remained constant at this height till the heart recommenced beating. It then rose to 76, 62, 53, 53, and on cessation of the stimulation, rose almost at once to 98 mm. i.e. rather higher than it had been before the excitation.

The behaviour of the venous pressures is more interesting. A rise of venous pressure is a familiar effect of vagus inhibition. It is not however so generally known that two distinct periods may be observed in the curve of venous pressure during the cardiac standstill.

Thus in Exp. 6 the various pressures before the excitation were

Portal Vein.	Inf. cava.
106	42

For the next 60 seconds the readings were

Portal vein.	Inf. cava.
88	
	74
96	
	81
98	

There is then a sudden rise in both manometers, the pressure in the inferior cava attaining the height of the portal pressure, and then rising with this latter. The readings were

Portal vein.	Vena cava.
	110
126	
	138
144	
	123

There is then a sudden drop in the vena cava pressure, directly the heart commences to beat, to 95, 60, and then 51 mm. The portal pressure, however, after a slight drop to 134, goes on rising, and attains its greatest height when the heart has recovered, and the arterial blood pressure is higher than it was at the commencement of the experiment. In this experiment the portal pressure went up to 204 mm. and then slowly descended to its original level, the descent occupying between two and three minutes.

A similar experiment is given in Protocol 10 (Diagram VI). One or two deviations which it presents from Exp. 6 are interesting as throwing some light on the nature of the factors involved. In this experiment, before excitation of the vagus, the pressures were

Fem. Art.	Portal vein.	Vena cava.
93	105	78

On excitation, the arterial pressure fell to 22, 12, 10, at which height it remained for 60 seconds. The portal pressure for the first 50 seconds was practically unaltered. The vena cava pressure gradually rose in the course of the first 50 seconds to 110, i.e., to the same height as the portal pressure. Ten seconds later, that is to say, one minute after the commencement of excitation, both venous pressures began to rise and in the course of another 70 seconds reached the height of 197 and 192 respectively. Just after the commencement of the large venous rise, the arterial pressure was noticed to be also rising slightly, and at the height of the venous rise, the arterial pressure had moved from 10 to 15 mm. Hg, a rise of 65 mm. $MgSO_4$. Excitation of the vagus was then discontinued after having lasted 150 seconds. The heart commenced to beat, but very slowly, and took about a minute and a half before it recovered its usual rhythm. We therefore got in the arterial pressure, first a sudden rise to 70 and then a slow gradual rise to 125. The course of the venous pressures on cessation of the excitation was essentially the same as in Exp. 6. In the present experiment, however, there was a distinct fall in the portal pressure to 137 before its second rise to 217.

The behaviour of the venous pressures on stimulation of the vagus is easily explicable in the light of the experiments we have already described. When the heart stands still in consequence of vagus stimulation, the only force driving the blood on is the elasticity of the distended arterial walls. This must cause a flow of blood into capillaries and veins, until the pressure is the same at all parts of the system.

Hence there is a fall of pressure in the arteries and a rise of pressure in the great veins near the heart, until in both regions the pressures are alike and equal to the mean pressure of the system. We see in both experiments a fall of arterial pressure to 10 mm. Hg, and a rise of vena cava pressure until it is equal to the portal pressure. The fact that the arterial pressure does not sink so low as the venous in the beginning of the experiment is due to the fact that under this low pressure, the blood is driven from the arteries into the veins extremely slowly, so that it takes some time for complete equilibrium to be established. The first part of the curves, viz. those obtained in the first 40 or 60 seconds after the commencement of the vagus excitation, simply show the effects of cardiac standstill and are exactly analogous to the curves obtained on stimulation of the vagus with divided cord. (Cp. Exp. 11 and Diagram I.) At the end of this period, a new factor makes itself felt. The cessation of the circulation, the anaemia of the brain, causes excitation of the vaso-motor centres, and active contraction of the smaller arteries, especially in the splanchnic area and the portal vein. The rise of peripheral resistance caused by this contraction does not here come into account, since here is no blood-flow through the vessels. What is important, however, is the diminution in the total capacity of the system caused by this constriction, in consequence of which the mean pressure of the system is increased and there must be a rise of pressure in all the vessels. Whether this rise will be seen in the arterial manometer depends on how far the equilibrium has been established before the anaemia has begun to affect the vaso-motor centre. The second experiment that we have quoted (Exp. 10) shows clearly that the rise affects arterial as well as venous pressure. We must regard the highest venous pressure reached during the cardiac inhibition as representing the mean general pressure of the vascular system, when the vaso-motor centre is acting strongly.

We have now to consider the second part of the curves, obtained after cessation of the stimulation. As the heart begins to pump blood from the big veins into the arteries, there is naturally a fall of vena cava pressure to its original height and a rise of arterial pressure. We showed above that on stimulating the splanchnics, the branches of the portal vein in the liver were constricted, and that this constriction outlasted the contraction of the arterioles, so that, on cessation of the stimulation, an increased supply of blood coming through the relaxing arterioles of the splanchnic area into the still contracted portal vein, caused a rise still higher of pressure in the latter vessel far exceeding

that which had been produced during the excitation itself. Exactly the same phenomenon is seen in these two curves (Diagrams V and VI). As the heart commences to beat, it takes up blood from the big veins and sends it on into the arteries and through them into the still constricted portal vein, so that the pressure in the portal vein is at its highest shortly after commencement of the vagal stimulation. It then gradually drops to its normal height as the branches of the portal vein gradually relax.

We have just stated that a large rise of venous pressure obtained when the heart is stopped by stimulation of the vagus, is chiefly conditioned by the active (asphyxia) contraction of the blood-vessels in the abdomen, i.e. those innervated by the splanchnic nerve. That this is the case is shown by the fact that if both splanchnics be divided, stimulation of the vagus no longer produces the customary venous rise. We get in fact, under these conditions, only the first phase of the normal vagus curve, so that the effects are almost identical, whether we divide the spinal cord in the neck, or merely divide both splanchnic nerves. Details of an experiment of this description are given in Exp. 1, and an example of the curves so obtained in Diagram VII. It will be noticed that the portal pressure is unaffected by the stimulation, while the vena cava pressure rises somewhat higher, until it is slightly above that of the portal.

Effect on capillary pressures.

We have shown above that on excitation of the vagus in animals with divided spinal cord, there will be a fall of pressure in the intestinal capillaries, while the pressure in the hepatic capillaries will be practically unchanged. Very different must be the effect in a normal animal. *During* the stimulation, while the heart is at a standstill, there is a large rise of pressure both in portal vein and in vena cava. Thus in Exp. 10 the pressure in the portal vein rose from 105 to 203 mm. $MgSO_4$, and in the vena cava from 78 to 192 mm. There must be therefore a corresponding rise of pressure in the hepatic capillaries. With regard to the intestinal capillaries it is difficult to decide, since there is a fall in arterial pressure coincident with the rise in portal pressure. All we can say is that the pressure in the intestinal vessels must be greater than when the cord is cut; but whether it is greater or less than normal we see no means of deciding.

When the heart recommences beating, the arterial pressure rises to

normal or above normal, while the portal vein pressure is largely increased. Hence immediately after the excitation is discontinued there will be a temporary rise of pressure in the intestinal capillaries.

During this period the pressure in the hepatic capillaries is probably about normal, for the vena cava pressure is normal, while the increased portal pressure is due largely to the increased resistance to the flow of blood through the liver.

Thus we find a great difference in the effect of vagus stimulation on the hepatic capillary pressure, depending on the presence or absence of the normal vaso-motor paths. This difference may perhaps be made clearer with the aid of a diagram.

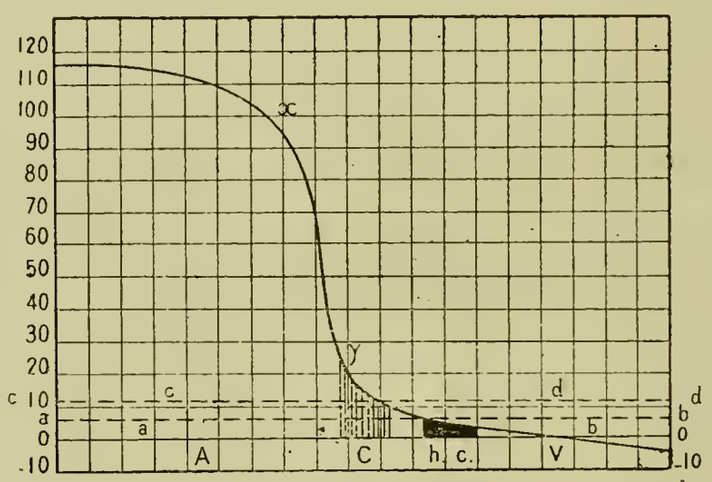


Fig. 3.

Fig. 3 represents the ordinary curve of the fall of blood pressure in the different parts of the vascular system. In this the largest fall of pressure is represented as taking place in the smaller arteries (from *x* to *y*). At *C* are the ordinary systemic capillaries. At *h. c.* are the hepatic capillaries, the level at *h* corresponding to the pressure in the portal vein. Now we may draw a line *ab* about 8 mm. Hg above the base line representing the general mean pressure, and therefore the pressure in any part of the system when no circulation is going on. If the cord be now cut, the mean pressure sinks in consequence of the general vascular dilatation, and would be a little below the line *ab*, and would in fact correspond very nearly to the hepatic capillary pressure. We see then that stimulation of the vagus in animals with cord cut (or splanchnics divided) will cause no rise in the hepatic capillary pressure, since the pressure cannot become more than the general mean pressure.

If however the vaso-motor centre is active, and there is general vascular constriction, the general mean pressure may be doubled and

may rise to a level represented by the line *cd*. If now the vagus be excited, and the circulation brought to a standstill, there must be a rise of pressure in the hepatic capillaries, from *ab* to *cd*, a rise of about 8 mm. Hg. We have dwelt rather fully on this point, since the liver is a very important source of the lymph which flows from the thoracic duct, and it is therefore necessary to understand the circumstances affecting capillary pressures in this organ.

Section II. Hydraemic Plethora and Hydraemia.

Hydraemic Plethora.

Although there are numerous researches dealing with the behaviour of the arterial blood pressure, after injection of large quantities of normal saline fluid into the circulation, yet in hardly any has the venous pressure been observed, although the amount of distension of and pressure in the veins under these conditions is of extreme importance, and is discussed freely by the various authors who have dealt with this subject. Cohnheim and Lichtheim[1], in their paper "Ueber Hydraemie und hydraemisches Oedem" describe a temporary rise of venous pressure accompanying the injection of the fluid, and lasting a short time after the injection. In their experiments, they measured the pressure in the external jugular vein by means of a soda manometer, but as they give no details of their experiments and the times of their various procedures are not mentioned at all, it is impossible to criticise them. Worm Müller, in his experiments on plethora, recognised the importance of an estimation of venous pressures, but regarded the means at his disposal as insufficient for this purpose and sought to determine the distribution of the injected blood in the body and the condition of the veins by inspection of the abdominal organs after death, and by observing the effect of pressure in the abdomen on the arterial blood pressure. Worm-Müller found that passive movements of the extremities, or pressure on the abdomen, after the production of artificial plethora, had very little effect on the arterial pressure, and argues from this that there cannot be any overfilling of the great veins. In answer to this we cannot do better than quote Johansson and Tigerstedt's criticism of the same. These authors remark "we believe, that in spite of this, there may be a distension of the veins of the abdomen. For, when all the great veins are overfull,

[1] *Virchow's Archiv*, Bd. xcvi. S. 106, 1877. Cp. also Lesser, *Ludwig's Arbeiten*, 1874.

an increased flow of blood to the heart brought about by pressure on the abdomen can have no effect on the cardiac activity, since the right heart has already enough and more than enough blood at its disposal."

We give at the end of this paper protocols of two experiments (Exp. 8 and 14), showing the result on venous and arterial pressures of injecting a large amount of salt solution into the circulation. The course of the pressure curves in Exp. 8 is shown in Diagram VIII. In this experiment the arterial pressure, which was 102 mm. to start with, rose gradually during the injection to 124, at which height it remained during the whole of the observation (22 min.). This behaviour of the arterial pressure on injection of quantities of fluid (i.e. a slight initial rise from 102 to 120, followed by no further rise however much fluid is injected) is typical, and has been observed by all workers on the subject since Worm-Müller.

Very different is the behaviour of the venous pressures. In Exp. 8 the injection of 500 c.c. normal saline lasted 7 min. During this time the portal pressure rose from 98 mm. $MgSO_4$ to 290 mm. and the vena cava pressure from 33 mm. to 225 mm. When the injection was discontinued, the portal pressure, after a further rise to 320, began to fall slowly, but 15 min. after the injection had been discontinued, was still 194 mm., that is to say, double as much as it was at the commencement of the experiment. At the end of the injection the vena cava pressure fell fairly rapidly, so that in 4 min. it had fallen from 225 to 115 mm. At this height it remained nearly stationary so that at the end of another 10 min. it still amounted to 120 m.

In Exp. 14, 350 c.c. of normal saline were injected during the space of $2\frac{1}{2}$ min. into a dog, which was already very hydraemic. During this time the arterial pressure rose from 60 to 70, the portal pressure from 87 to 167, and the vena cava pressure from 34 to 124. The further course of the experiment was very similar to that observed in Exp. 8. Five minutes after the end of the injection the pressures were

Fem. Art.	Portal Vein.	Vena Cava.
72 mm. Hg	187 mm. $MgSO_4$	74 mm. $MgSO_4$

Ten minutes later the pressures were:

Fem. Art.	Portal Vein.	Vena Cava.
72	110	50

Half an hour later the pressures were nearly normal, the arterial pressure however being higher than at the commencement of the experiment.

It is evident from these experiments that Cohnheim undervalued the part played by the veins in the adaptation of the circulation to the injection of larger quantities of fluid. It is true that in these experiments the rise of venous pressure was not absolutely permanent. It lasted however 20 to 30 min. after the end of the injection, during which a large quantity had had time to leave the vessels either as lymph or increased secretions (salivary glands, kidneys, &c.). In another experiment in which a dog was injected with 350 c.c. of defibrinated blood from another dog, the pressures in the vena cava and portal vein were still increased 40 min. after the termination of the injection.

The first effect of the injection of fluid into the circulation is a rise of pressure in all parts of the system, arterial and venous. After the first small rise, the arterial pressure becomes constant, and room is found for the fluid subsequently injected by the active dilatation of the arterioles, so that there is a constantly diminishing difference between arterial and venous pressures. The pressure in the veins therefore goes on rising during the whole time that fluid is being injected. By this rise of pressure the thin walls of the veins are stretched, and the total capacity of the system largely increased. The fall of venous pressure on discontinuing the injection is probably due partly to the increased stretching of the walls of the veins under the abnormal pressure to which they are subjected, and largely to the escape of the injected fluid through the vessel walls.

Under these conditions, so long as the heart can perform the increased work which is put upon it, the velocity of the blood in the vessels must be largely increased, since the augmented pressure in the large veins provides a larger flow of blood into the heart, and the peripheral resistance is lowered by the relaxation of the arterioles. Cohnheim has in fact demonstrated that the velocity of the blood in plethora is largely increased.

Effect on capillary pressures.

During and for 20 to 30 minutes after injection, the arterial pressure is increased, the portal pressure is largely increased, and the arterioles are relaxed, all three factors combining to cause great increase of pressure in the capillaries of the intestines.

In the hepatic capillaries too, there must be a large increase of pressure. In Exp. 8, the pressure in the portal vein rose from 98 to a maximum of 320 mm. and then fell slowly to 194 mm. The vena cava

pressure rose from 33 to 245 mm. and then fell, quickly at first and then more slowly, to 120 mm. There is then a large rise of pressure on both sides, afferent and efferent, of the hepatic capillaries, and there must be also a corresponding rise in the capillaries. The increase in the difference between the portal and vena cava pressures shows us that the velocity of the blood current through the liver must also be very largely increased, so that, although we have increased venous pressures here, the hyperaemia is active, not passive; and increased venous pressure does not necessarily involve venous congestion or 'Stauung' as Cohnheim[1] seems to imply. The direct measurement of the pressures in the portal vein and vena cava simply bear out what has been often described as a result of plethora or hydraemic plethora, viz., a swelling of the liver, which is extremely full of blood, so that, on section, a large amount of fluid runs away from it. This condition is well described by Johansson and Tigerstedt[2]. They say "the second fact to which we would draw attention is the great distension of the liver which we have observed in all our transfusion experiments and which has been also remarked by earlier workers. After the injection of large quantities of fluid the liver becomes almost as hard as a board. If after the death of the animal, the liver be cut out, fluid streams from it in great quantities. We see then that a considerable quantity of fluid is taken up by the liver and thus withdrawn from the general circulation."

Simple hydraemia.

The effects of simple hydraemia as shown in Exp. 13 and Diagram X. In this experiment the animal, which weighed about 7 kilos, was bled to 200 c.c. from the right femoral artery. Before the bleeding the pressures were:

Fem. Art.	Portal Vein.
77 mm. Hg	103 $MgSO_4$

The vena cava pressure could unfortunately not be ascertained, owing to faulty position of the femoral vein cannula. The bleeding lasted one min. Seventy-five secs. after the bleeding the pressures were:

Fem. Art.	Portal Vein.
54	42

[1] *Gesammelte Abhandlungen*, p. 574.

[2] "Gegenseitige Beziehungen des Herzens und der Gefässe." *Skand. Archiv f. Phys.* Vol. I. p. 396, 1889.

200 c.c. normal saline were allowed to flow into the right external jugular vein. The injection lasted 2 min. At the end of the injection the pressures were:

Fem. Art.	Portal Vein.
68	147

and three min. later

80	147

The rise of pressure in the portal vein was only transitory. Ten minutes later, when the position of the fem. vein cannula had been rectified, the portal pressure had fallen to 87. A second quantity of blood (150 c.c.) was then drawn from the femoral artery. Before the bleeding the pressures were:

Fem. Art.	Portal Vein.	Vena Cava.
74	90	38

At the end of the bleeding they were:

Fem. Art.	Portal Vein.	Vena Cava.
26	45	22

150 c.c. of normal saline were then injected. At the end of the injection the pressures were

Fem. Art.	Portal Vein.	Vena Cava.
52	77	36

and three minutes later,

Fem. Art.	Portal Vein.	Vena Cava.
62	77	28

We see that simple hydraemia has very little effect on the pressures, there being however a small general fall both in arterial and venous pressures. The large rise in the portal pressure which is obtained after the first injection of normal saline, is probably due to the fact that the injection was carried out more rapidly than the vascular system could accommodate the access of fluid, since it was only transitory, and disappeared entirely at the end of a few minutes. In the same way there will be very little effect on capillary pressures. The first injection of fluid may be followed by a small rise in capillary pressure in the intestinal vessels, but the rise is only transitory.

This experiment also illustrates the effects on the general pressures of diminishing the total quantity of circulating fluid. As we might expect, bleeding gives rise to a fall of pressure in all parts of the system, in the fem. art., in the portal vein and vena cava, and it will be

observed that after the first bleeding in this experiment, the gradual recovery of the arterial pressure took place at the expense of the venous pressure. All the reactions of the vaso-motor system seem to be directed towards maintaining the pressure in the large arteries at a constant height. If the amount of blood in the body is diminished, the veins become less distended, in order to supply more blood to the arteries. If there is a hyper-normal amount of fluid in circulation, nearly the whole excess is held by the distended veins and capillaries, the tension and therefore the amount of blood in the larger arteries remaining nearly constant.

Section III.

Mechanical obstruction of the larger blood vessels.

Obstruction of the thoracic aorta.

In these experiments the thoracic aorta was obstructed above the diaphragm by distending a rubber obturator which had been introduced through the external iliac artery[1]. The course of the pressure curves in the femoral artery, and portal and femoral veins may be seen in Exp. 7. In Exp. 7, the pressures at the commmencement of the experiment were

Fem. Art.	Portal vein.	Fem. vein.
82	76	43

Half a minute after the aorta was obstructed, the pressures were

Fem. Art.	Portal vein.	Fem. vein.
12	48	41

So that there is a simultaneous fall of pressure in the arteries and in the portal vein, while the pressure in the peripheral end of the vena cava was unaltered.

In some earlier experiments, when estimating the vena cava pressure alone, we had found a slight rise of pressure in this vessel on obstruction of the thoracic aorta. Thus in an experiment on June 26th, the vena cava pressure before obstruction was 59. On obstructing the aorta, it rose to 69, and then gradually fell to 63. On releasing the

[1] We have recently repeated this experiment in a large dog, in which we introduced the aortic obturator through the right carotid artery. The results however were exactly similar to those obtained when the obturator was introduced in the way described above.

obstruction, it fell at once to 57. In another observation, the pressure before obstruction was 63, after obstruction it rose to 72, and then slowly fell to 66. These rises are so slight that we may say that the pressure in the central end of the femoral vein is practically unaltered.

With regard to the effect of the obstruction of the aorta on capillary pressures, it is evident that the pressure in the intestinal capillaries must be very much reduced, since there is a large fall of pressure both in the arterial and venous side of these vessels. The effect on the capillary pressure in the liver is not quite so certain, for although we find the pressure in the peripheral end of the vena cava practically unaltered, yet it must be remembered that the pressure at this point is always higher than the pressure at the point of opening of the hepatic veins, the difference in the pressures being proportional to the velocity of the blood flowing from the external iliac to the hepatic veins. By obstruction of the aorta, we reduce the velocity of this blood current to a minimum, and therefore the difference between the femoral venous pressure and the hepatic venous pressure must also be largely reduced. Hence the fact that the pressure in the central end of the femoral vein is unaltered or slightly increased must imply a definite and probably considerable rise of pressure in the hepatic veins, so that there is a fall of pressure on the portal vein side, and a rise of pressure in the hepatic vein side of the capillaries in the liver. It thus becomes a matter of speculation what change really does take place in the hepatic capillary pressure. It may be diminished, but having regard to the greater dependence of capillary on venous than on arterial pressures, it is, we think, possibly unaltered or somewhat increased.

Obstruction of inferior vena cava above diaphragm.

The effect of obstruction of the inferior vena cava above the diaphragm is shown in Exp. 2, and graphically in Diagram VIII. In all cases, the vena cava was obstructed by means of an obturator introduced through the right external jugular vein. In Exp. 2, the pressures at the commencement of the experiment were

Fem. Art.	Portal vein.	Vena cava.
72	89	51

On complete obstruction of the vena cava, the pressure rose rapidly in both venous manometers to 240 mm. $MgSO_4$, while the arterial

pressure sank to 36. All three pressures then gradually sank, so that, after the obstruction had lasted 4 minutes, the pressures were

Fem. Art.	Portal vein.	Vena cava.
26	226	212

It will be seen that, although the pressures at the beginning of the obstruction are the same in both portal vein and vena cava, there is soon established a slight difference in favour of the portal vein, showing probably that there is still a slow current of blood passing through the liver. The gradual fall in all three pressures during the maintenance of the obstruction is, we take it, due partly to the increased transudation of fluid and partly to the gradual stretching of the walls of the vessels, especially the veins, an occurrence to which we referred earlier in this paper, and to which Worm-Müller ascribed the fact that an artificially plethoric dog might be bled to death, while his vessels still contained more than their normal quantity of blood.

On letting the water out of the obturator, there is at once a sudden fall in both venous pressures, and a rise in the arterial pressure to its former height.

Effect on capillary pressures. In Exp. 2, the pressure in the portal vein during the obstruction was nearly trebled, and that in the vena cava was more than quadrupled. There must therefore be a corresponding rise of the pressure in the hepatic capillaries to three or four times its normal height. This fact explains the extreme congestion and swelling of the liver which is observed if the animal be killed or dies while the vena cava is still obstructed. It is impossible to say in what way the pressure in the intestinal capillaries is affected. It is true, as Heidenhain has pointed out, that during the obstruction the intestines look blanched and anaemic. But we have already shown that apparent anaemia of a part does not necessarily imply diminished capillary pressure in that part, but may, indeed, be accompanied by increased pressure. The arterial and venous pressures move in different directions, the arterial pressure falls from 72 to 26 mm. Hg, while the portal vein pressure rises from 90 to 226 mm. $MgSO_4$, i.e., from 7 to 17 mm. Hg. The factors at our disposal do not enable us to decide in what direction the pressure in the intestinal capillaries is affected by the obstruction of the inferior vena cava above the diaphragm.

In Exp. 7 we investigated the influence of obstruction of the inferior cava when the thoracic aorta was already obstructed. As might be expected, we get a slow steady rise in both venous mano-

meters, but the slowness of the rise bears witness to the very small amount of blood that is entering the lower part of the aorta. Four minutes after the obstruction of the inferior cava, the pressure in the two veins had only reached 105 mm. $MgSO_4$, in the last two minutes of which it had only risen 5 mm. Under these circumstances the flow of blood through the abdominal organs must be practically at a standstill, as is shown by the equality of the two venous pressures. On releasing the aorta, we at once obtained the usual large rise of pressure in the two venous manometers, the portal pressure rising to 280 mm. and the vena cava pressure to 235 mm. $MgSO_4$. It is worth noticing that in this case the vena cava pressure rose higher than the pressure in the portal vein, an occurrence probably due to the fact that the obturator was immediately over the opening of the hepatic veins.

Obstruction of the portal vein.

On obstruction of the portal vein the rise of pressure produced in this vein was too great to be recorded by our manometers. We found in other experiments that this rise of pressure might amount to 600 or 800 mm. $MgSO_4$. The only point of interest about this experiment is the behaviour of the arterial pressure on obstruction of the portal vein. It will be noticed that obstruction of the portal vein causes a rise of arterial pressure (in one observation from 92 to 102 mm. Hg), and that the release of the ligatured portal vein brings about a temporary fall (from 104 to 82 mm.). These effects must be due to the increase or diminution of the resistance of the splanchnic area caused by ligature or release of the portal vein. The familiar fall of arterial pressure which follows ligature of the portal vein is a later phenomenon, and does not make its appearance until one or two minutes have elapsed after the ligature has been applied. We might perhaps ascribe the rise of pressure on ligature of the vein to the sudden cutting off of the large capillary area of the liver, but the two factors (diminished area, and increased resistance) are so intimately connected, that the difference between them is more one of description than of fact.

Conclusion.

Our results then confirm in the main the principles of the circulation which have been so ably worked out by Ludwig and his pupils, and even those results which are new only afford further

examples of the universal applicability of these principles. Of these we may especially draw attention to the existence of two phases in the rise of venous pressure after vagus stimulation, and to the importance of the rise of venous pressure in plethora and hydraemic plethora.

It may be useful to summarise here our conclusions with regard to the effect of the various procedures employed in the capillary pressures in the abdominal organs.

Procedure employed	Capillary pressure	
	Intestines &c.	Liver
Section of cord	Fall	Slight fall
Stimulation of vagus. Cord cut	Large fall	Slight rise
Stimulation of vagus. Splanchnics cut.	Large fall	Slight rise
Stimulation of vagus. Normal	Probable fall during excitation	Rise during excitation
	Rise after excitation	Normal after excitation
Asphyxia, normal animal	Slight rise (?) then a gradual fall	Rise till death
Asphyxia with cord or splanchnics cut	Fall	? Probably unaltered
Excitation of splanchnics	Rise after excitation	Slight rise during excitation (?)
Anaemia	Fall	Fall
Plethora and hydraemic plethora	Large rise	Large rise
Simple hydraemia	Unaffected	Unaffected
Obstruction of aorta	Large fall	Doubtful. Perhaps unaffected or a small rise?
Obstruction of inferior vena cava above diaphragm	Doubtful. Perhaps unaffected	Very large rise
Obstruction of portal vein	Very large rise	Fall

It will be seen from these results how fallacious it is to argue that a fall of arterial blood pressure necessarily implies a fall of pressure in all the capillary areas of the body. Short of a direct determination of the pressure in the capillaries (for which we have not yet a sufficiently reliable or convenient method) we can only judge of changes in the capillary pressure when we take the pressures on both sides of the capillary area, viz. in both afferent and efferent vessels. Our results will serve to illustrate both the applications and the limitations of this method.

Note. We have mentioned that the fall of arterial pressure which is produced by section of the cord is conditioned by two factors, viz.:

(*a*) Lowered peripheral resistance,

(*b*) Increased capacity of the whole vascular system.

We have lately sought to obtain some conception of the part played by each factor in the full, and although our method is not free from objections, we think the experiment is worth quoting.

The second factor (increased capacity) causes the fall in arterial pressure immediately by diminishing the flow of blood from the great veins into the heart. We thought then that we might neutralise this factor by injecting warm defibrinated blood into veins of a dog after section of the cord, until the pressures in the portal vein and vena cava were equal to their former values before the cord was divided. The pressure in the great veins being thus restored to normal, the heart would have the same amount of blood at its disposal, and if its powers were unchanged, would drive the normal amount of blood into the aorta at each beat, so that now the only factor tending to diminish the arterial blood pressure would be the lessened peripheral resistance.

We give here the details of the experiment:

Oct. 18, 1893.

Dog 6·3 kilos. Cannulae in left fem. art., in splenic vein and in left external iliac vein. Spinal cord exposed about 1st dorsal vertebra.

Time	Fem. Art.	Portal Vein	Vena Cava
3·35	106	100	16
	106	98	14
	107	93	12
	107	92	12
3·43	Cord divided at 1st dorsal vertebra		
4·14	58	57	4
	56	57	4
4·16	20 c.cm. blood injected		
4·17	64	?	10
4·37	30 c.cm. more blood injected		
4·41	25 c.cm. more blood run in		
4·42	70	78	14
	71	78	10
4·43	25 c.cm. blood injected		
4·47	84	88	8
	82	85	8
4·48	Another 25 c.cm. blood injected		
4·51	96	109	16
	96	108	17
	95	110	18

Dog weighed 6·3 kilos. Total amount of defibrinated blood injected = 125 c.cm.

It will be seen from this that the normal venous pressures were not reached until between 100 and 125 c.cm. of defibrinated blood had been injected. Now this dog had almost 500 c.cm. of blood in his body, so that, if our arguments are correct, the dilatation of vessels ensuing on section of the cord at the 1st dorsal vertebra increases the capacity of the system by 125 c.cm., i.e. by a quarter of its whole capacity.

Mall has shown that on stimulation of the splanchnic nerves the amount of blood driven out of the constricted vessels may amount to from 3 to 27 per cent. of the total blood of the body, and our experiment seems to show that as great a change in the opposite direction in the capacity of the abdominal and other vessels is produced by their complete relaxation. It is interesting moreover to notice that after the restoration of the venous pressures, the arterial pressure is not far below the point at which it stood before the division of the cord, and is in fact only 10 mm. Hg lower.

From this one experiment, then, we might say

Total fall of arterial pressure produced by section of the cord = 50 mm.

Of this fall 10 mm. is due to the diminished peripheral resistance, and 40 mm. to the increased capacity of the whole system, a conclusion which we were extremely surprised at. We have not continued the investigation of this subject, since there is one possible fallacy in our method: viz. the impossibility of being sure that the amount of fluid driven out by the heart is really restored to the normal. The obvious way of getting over this difficulty would be to use, not the venous pressures, but the "second volume" of the heart as the criterion of the amount of fluid to be injected. Probably the use of an instrument such as Roy's cardiometer would be the best means of solving this difficulty.

DESCRIPTION OF DIAGRAMS.

In all the diagrams, the divisions along the ordinates represent 10 mm. The arterial pressures are expressed in millimetres of mercury, the venous pressures in millimetres of 25 $^{0}/_{0}$ solution of magnesium sulphate in water.

The thick line is the pressure in the femoral artery.

The broken line is the pressure in the central end of the common femoral vein (inf. vena cava pressure).

The thin line is the pressure in the portal vein, as registered by a manometer connected with the splenic vein.

The divisions along the abscissa correspond in most cases to 10 second intervals. In VIII. and IX. however each division represents 30 seconds.

Diagram I. (Protocol 11).
Excitation of vagus in a dog in which the spinal cord had been divided in upper dorsal region.

Diagram II. (Protocol 5).
Excitation of both splanchnic nerves.

Diagram III. (Protocol 4).
Asphyxia. Normal animal.

Diagram IV. (Protocol 12).
Asphyxia in dog with divided cord.

Diagram V. (Protocol 3).
Excitation of vagus. Normal animal.

Diagram VI. (Protocol 10).
Excitation of vagus. Normal animal.

Diagram VII. (Protocol 1).
Excitation of vagus. Both splanchnics divided.

Diagram VIII. (Protocol 2).
Obstruction of inferior vena cava above diaphragm.

Diagram IX. (Protocol 8).
Hydraemic plethora.

Diagram X. (Protocol 13).
Simple hydraemia.

PROTOCOLS.

In each of these experiments the figures in the right-hand column follow on those of the left, whether the latter are on one page or on two.

Experiment 1.

Excitation of vagus. Splanchnics divided.

July 13, 1893. Dog about 8 kilos. Both large splanchnics divided. Smaller splanchnics intact (?), at any rate not cut. Left femoral artery to Hg manometer. Left femoral vein and splenic vein to $MgSO_4$ manometers (25 %). Reading of arterial pressure every 10 seconds. Right vagus divided on Ludwig electrodes. Left vagus intact.

	$MgSO_4$	
Art. Hg	Portal Vein	Inf. Cava
101	65	
101		42
101	65	
101		40
101	64	
101		40

	$MgSO_4$	
Art. Hg	Portal Vein	Inf. Cava
40		76
14	61	
	Six beats.	
66		48
71	56[1]	
	Excitation stopped.	

101	63	
101		38
101	63	
101		37
	Vagus excited.	
18	60	
13		72
12	61	
11		79
11	61	
	One beat.	
72		51[2]
74	56	[2]
82		48[3]
86	65	
88		51
91	69	
92		50
94	69	
96		48
96	66	
97		—

[1] Heart beating slowly. [2] Heart still slow. [3] Pulse rate normal.

Experiment 2.

Obstruction of inf. cava above diaphragm.

July 19, 1893. Cannulae in fem. art. and inf. cava and portal vein. Left vagus divided and on electrodes. Obturator in inf. cava (splanchnics intact).

Hg man.	$MgSO_4$ man.	
Fem. Art.	Portal Vein	Inf. Cava
64	87	
66		52
69	89	
72		51
71	90	
73[1]		—
	One syringeful injected.	
62	—	
62		—
	Second syringeful[2].	
42		240
36	240	
32		240
31	242	
30		232
30	241	
30		231
30	242	
28		231
28	240	
28		228
28	238	
27		226
27	238	

	$MgSO_4$ man.	
Fem. Art.	Portal Vein	Inf. Cava
27		221
27	234	
27		220
27	230	
27		217
27	228	
27		215
27	228	
27		213
26	226	
26		212
26		
	Water let out of obturator.	
64		42
70	80	
66		46
65	100	
66		46
68	101	
71		46
73	102	
76		45
	Obturator then removed altogether.	

[1] No reading, [2] Complete obstruction.

Experiment 3.

Stimulation of vagus. Normal animal.

July 19, 1893. (Same experiment continued.) Left vagus on Ludwig electrodes.

Hg man.	$MgSO_4$ man.	
Fem. Art.	Portal Vein	Inf. Cava
87		44
88	100	
88		43
90	104	
90		43
90	106	
90		42
Vagus excited. Coil 5 cm.		
26	88	
20		74
16	96	
10		81
10	98	
10		110
10	126	
11		138
76	144	[1]
62		123[1]
53	134	[1]
53		95[1]
Vagus off.		
84	154	
98		60

Hg man.	$MgSO_4$ man.	
Fem. Art.	Portal Vein	Inf. Cava
95	204	
92		54
93	180	
93		51
94	170	
94		51
94	158	
94		49
94	140	
94		49
94	127	
94		49
94	116	
94		47
94	108	
94		44
94	106	
96		42
96	100	
97		42
97	100	
98		46

[1] Beating slowly.

Experiment 4.

Asphyxia. Both vagi cut. Splanchnics intact.

July 19, 1893. (Same experiment continued.)

Hg man.	$MgSO_4$ man.	
Fem. Art.	Portal Vein	Inf. Cava
126	90	
126		48
122	84	
120		48
118[1]	—	
118[1]		—
116[1]	—	
115[1]		—
128[1]	—	

Hg man.	$MgSO_4$ man.	
Fem. Art.	Portal Vein	Inf. Cava
53	142	
50		145
45	134	
40		136
36	131	
29		134
24	127	
21		128
18	122	

134	124	[2]	15		124
139		170[2]	13	118	
136	124	[2]	12		120
135		70[2]	12	114	
137	114	[2]	12		116
120		78	12	110	
94	120		11		112
87		96	11	104	
80	128		10		106
84		106	10	101	
84	136		10		100
81		134	10	98	
79	142		10		98
78		140	10	96	
69	142		10		
65		140	10		96
61	144		9	92	
58		144	9[3]		
56		144			

[1] No readings. Both pleural cavities opened.

[2] Strong respiratory movements, causing enormous excursions of venous manometers.

[3] At 1 h. 30 m. At 1 h. 45 m. pressures were:

Fem. art. 5 mm. Hg. Portal vein 59 mm. $MgSO_4$.
Inf. cava 58 mm. ,,

Experiment 5.

Excitation of both splanchnics. (*One vagus intact.*)

July 20, 1893.

Fem. Art.	Portal Vein	Inf. Cava	Fem. Art.	Portal Vein	Inf. Cava
93	92		116		68
93		82	108	190	
94	92		104		64
92		72	99	186	
94	92		96		64
94		68	90	164	
	Splanchnics excited.		88		66
111	92		88	140	
116		84	90		66
111	105[1]		93	120	
118		86	96		68
121	140[1]		98	106	
124		86	98		68
125	170[1]		96	104	
126		82	102		68
129	160[1]		96	104	
132		84	102		68
132	160[1]		96	104	
132		82	101		66
122	180		102	102	
	Excitation stopped.			Right vagus was then cut.	

[1] Heart slowed.

Experiment 6.

Asphyxia. Splanchnics and vagi cut. Excitation of splanchnics.
July 20, 1893. (Same experiment continued.)

Fem. Art.	Portal Vein	Inf. Cava
60	102	
60		67
58	100	
57		65
56	97	
55		65
54	96	
54[1]	—	—
48[1]	—	—
49[1]	—	—
50[1]	—	—
48[1]	—	—
46[1]	—	—
42	120[2]	
42		63
38	94[3]	
37		58
37	90	
38		58
38	88	
36		58
34	84	
31		62
28	83	
25		68
	Splanchnics excited.	
25	94	
31		84
30	108	
29		90
26	110	
25		98
21	108	

Fem. Art.	Portal Vein	Inf. Cava
20		102
	Excitation off.	
17	106	
16		98
14	95	
12		90
11	88	
10		88
10	84	
10		84
	Splanchnics excited.	
10	86	
10		88
10	93	
10		90
10	95	
10		90
	Excitation off.	
10	93	
10		88
10	82	
10		82
9	77	
9		78
9	75	
9		76
9	73	
9		74
9	72	
9		72
9	70	
9		72[4]
6	48	50[5]

[1] No readings. Both pleural cavities opened.
[2] Strong respiratory movements.
[3] Heart slower.
[4] At 2 h. 10 m.
[5] At 2 h. 55 m.

Experiment 7.

Obstruction of aorta with obstruction of inf. cava.
July 21, 1893.

Fem. Art.	Portal Vein	Inf. Cava
80		43
80	74	
80		43
86	76	
82		43
	Aorta obstructed.	
36		47
18	54	
14		43
13	48	
12		45
12	48	
12		41
12	48	
12		41
12	48	
12[1]	—	—
12[1]	—	—
12[1]	—	—
12[1]	—	—
12[1]	—	—
12	68	
13		70
13	72	
13		75
13	78	
13		80
14	84	
14		87
14	88	
14		91
14	94	
14		95
14	98	
13		100
13	98	
13		100
13	102	
13		101
13	104	
13		103
13	104	

Fem. Art.	Portal Vein	Inf. Cava
13		103
13	106	
13		105
13	—	
	Water let out of aortic balloon.	
42	138	
35		285
35	248	
34		325
34	268	
34		365
34	260	
34		325
34	280	
34		335
	Water let out of vein obturator.	
71	130	
76		45
66	100	
66		57
66	112	
67		61
70	112	
72		61
72	108	
74		59
76	104	
78	104	
80		53
85	94	
84		51
85	90	
88		51
88	88	
88		47
88	84	
90		47
88	82	
90		47
90	80	
90		45

[1] Vena cava obstructed. No readings.

Experiment 8.

Hydraemic plethora.

July 24, 1893. A cannula in rt. extl. jug. vein for running in normal saline. Readings every 30 seconds.

Fem. Art.	Portal Vein	Inf. Cava
102	98	33
Injection of 500 c.cm. normal saline begun.		
110	120	
100		81
98	200	
102		125
100	230	
106		175
110	250	
112		185
120	270	
122		225
121	280	
127		245
122	290	
124		225
Injection finished.		
126	320	
128		185
124	270	
126		145
126	260	
124		135
126	252	
124		115
125	244	
124		105
124	238	
123		100
123	234	
122		97
122	228	
128		101
122	222	
122		101
124	212	
122		103
123	198	
Pause of two minutes.		
122		117
122	194	
122		120
122	—	—

Experiment 9.

Bleeding (Anaemia).

July 24, 1893. (Same experiment continued.) A. C. E. given and cannula inserted in rt. extl. iliac artery for bleeding. Readings every 30 seconds.

Fem. Art.	Portal Vein	Inf. Cava
98		93
94	118	
Bleeding from iliac artery commenced.		
48	72	80
22	46	75
Bleeding stopped.		
12	54	80
30	52	77
28	52	73
Bleeding recommenced.		
12	58	73
12	58	83
10	60	85
8	60	89
8	48	80
9	45	71
9	45	68
9	44	65[1]
1	29	34[2]

[1] At 3 h. 44 m.

[2] At 4 h. 20 m.

Experiment 10.

Stimulation of Vagus. (Normal.)

July 25, 1893. Left fem. art. to Hg man. Portal vein and left inf. cava to $MgSO_4$ manometers. Rt. vagus intact. Left vagus cut and peripheral end on Ludwig electrodes. Cord exposed at lower border of 7th cervical vertebra (or 1st dorsal). Reading every 10 seconds.

Fem. Art.	Portal Vein	Inf. Cava
90	105	
92		78
92	106	
94		78
93	105	
	Vagus excited.	
22		84[1]
12	103	[1]
10		88[1]
10	107	[1]
10		110[1]
10	115	[1]
10		140[1]
10	153	[1]
11		170[1]
12	182	[1]
13		186[1]
14	203	[1]
15		192[1]
16	197	[1]
14		190[1]
	Excitation stopped.	
70	137	[2]
58		86[2]
56	157	[2]
56		92[2]
26	147[1]	
64		76[2]
64	183	[3]
68		68
80	217	
76		66
76	207	
80		64
82	181	
88		64
92	157	
96		64
98	141	
102		64
106	127	
108		64
112	117	
114	107	
116		64
118	105	
118		62
120	101	
122		60
122	97	
125		60
125	97	
	A. C. E. mixture given and the cord then divided above 1st dorsal vertebra.	

[1] Heart stopped. [2] Heart beating slowly.
[3] Heart beating regularly and gradually getting faster.

Experiment 11.

Stimulation of vagus. Cord divided.

July 25, 1893. Same experiment continued, but cord divided at 7th C. or 1st D. vert.

Fem. Art.	Portal Vein	Inf. Cava
	Cord cut.	
62	73	
62		61
59	73	
58		62
58	73	
58		62
	Vagus excited.	
7	82	
7		79
7	82	
$6\frac{1}{2}$		80
	Excitation stopped[1].	
$6\frac{1}{2}$	83	
$6\frac{1}{2}$		82
$6\frac{1}{2}$	83	

20	72	
13		68
11	77	
9		72
8	71	
7		72
7	72	
7		74
7	79	
7		76
7	80	
7		78
$6\frac{1}{2}$		80
$6\frac{1}{2}$	82	
$6\frac{1}{2}$		80
$6\frac{1}{2}$[2]	—	—
34[2]	—	—
40[2]	—	—
40[2]	—	—
40[2]	—	—
42		46[3]
42	81	
42		46
42	81	

[1] Heart does not beat.

[2] No readings. Heart not beating, so fingers introduced into abdomen and heart pinched rhythmically till it once more beat of itself. Rt. vagus cut.

[3] C. (About 2 min. later.) Heart beating spontaneously and regularly.

Experiment 12.

Asphyxia. Cord divided. Both vagi cut.

July 25, 1893. Same experiment continued. (Reading every 10 secs.)

Fem. Art.	Portal Vein	Inf. Cava
58	87	
59		48
60	89	
61		48
62	89	
63		48
Both pleural cavities opened.		
62	—	—
60	—	—
63		50
64	89	
65		50
60	91	
62		52
61	97[1]	
59		52
58	101	
54		52
49	105	
46		52
42	93	
40		48
36	87[2]	
36		50
34	81	
33		50
30	73	
28		50
26	71	
22		48
20	67	
18		52
16	67	
16		54
15	67	
14		56
12	66	
11		58
10	66	
10		60
10	66	
10		62
9	67	
9		64
9	67	
9	66	
9		64
9	65	
9		64
9	65	[3]
9		64[3]
	61	61
	Spinal cord pithed.	
	53	52[4]

[1] Quick respirations. [2] Respiration slower. [3] At 3 h. 4 m. [4] At 3 h. 53 m.

Experiment 13.

Hydraemia (readings every 30 secs.).

July 26, 1893. Left fem. art. to Hg man. Splenic vein and left fem. vein to $MgSO_4$ manometers. Cannulae in rt. ext. jugular for running in saline and in rt. fem. art. for bleeding.

Fem. Art.	Portal Vein
80	103
80	101
177	101
62	73[1]
42	53[1]
42	42
50	43
54	42
Saline injected.	
60	51[2]
72	61[2]
72	170[2]
64	150[2]
End of injection.	
68	147
69	137
78	132
76	147
82	147
80	147[3,4]
Valve in fem. vein got rid of.	

Fem. Art.	Portal Vein	Inf. Cava
78	87	
70	97	36
74	90	38
Bleeding begun.		
58	81	30
36	51	22
26	41	
Bleeding ended.		
26	45	22
Injection of 150 c.cm. saline.		
42	60	32
46	67	32
54	83	38
End of injection.		
52	77	36
56	75	36
58	72	30
60	71	28
63	71	26
62	77	28[5]

[1] Bled to 200 c.cm. [2] 200 c.cm. normal saline injected.
[3] A valve in femoral vein, so all femoral readings up to now had to be rejected.
[4] At 2 h. 56 m. [5] At 3 h. 14 m.

Experiment 14.

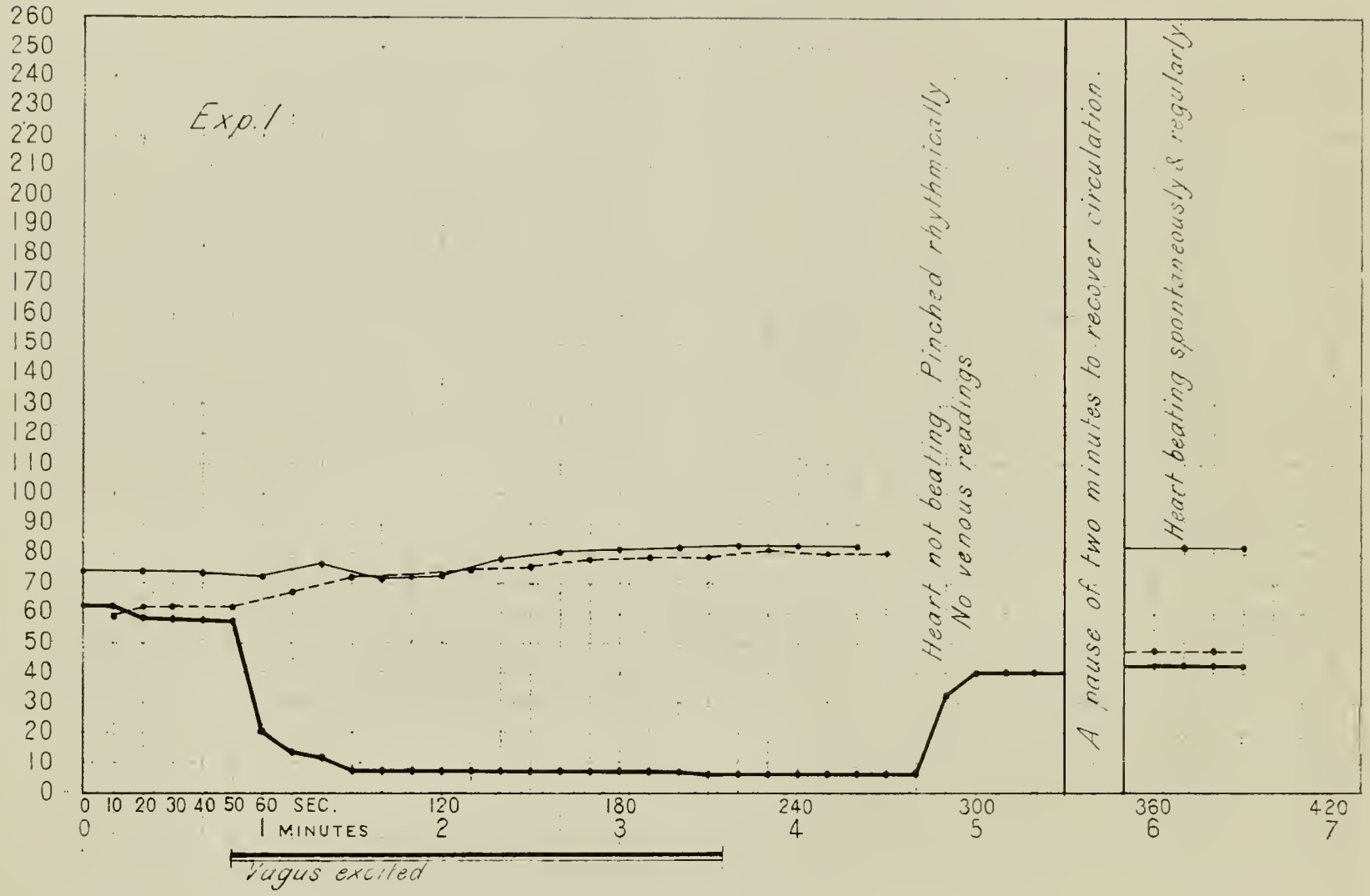
Hydraemic plethora.

July 26, 1893. (Same experiment.)

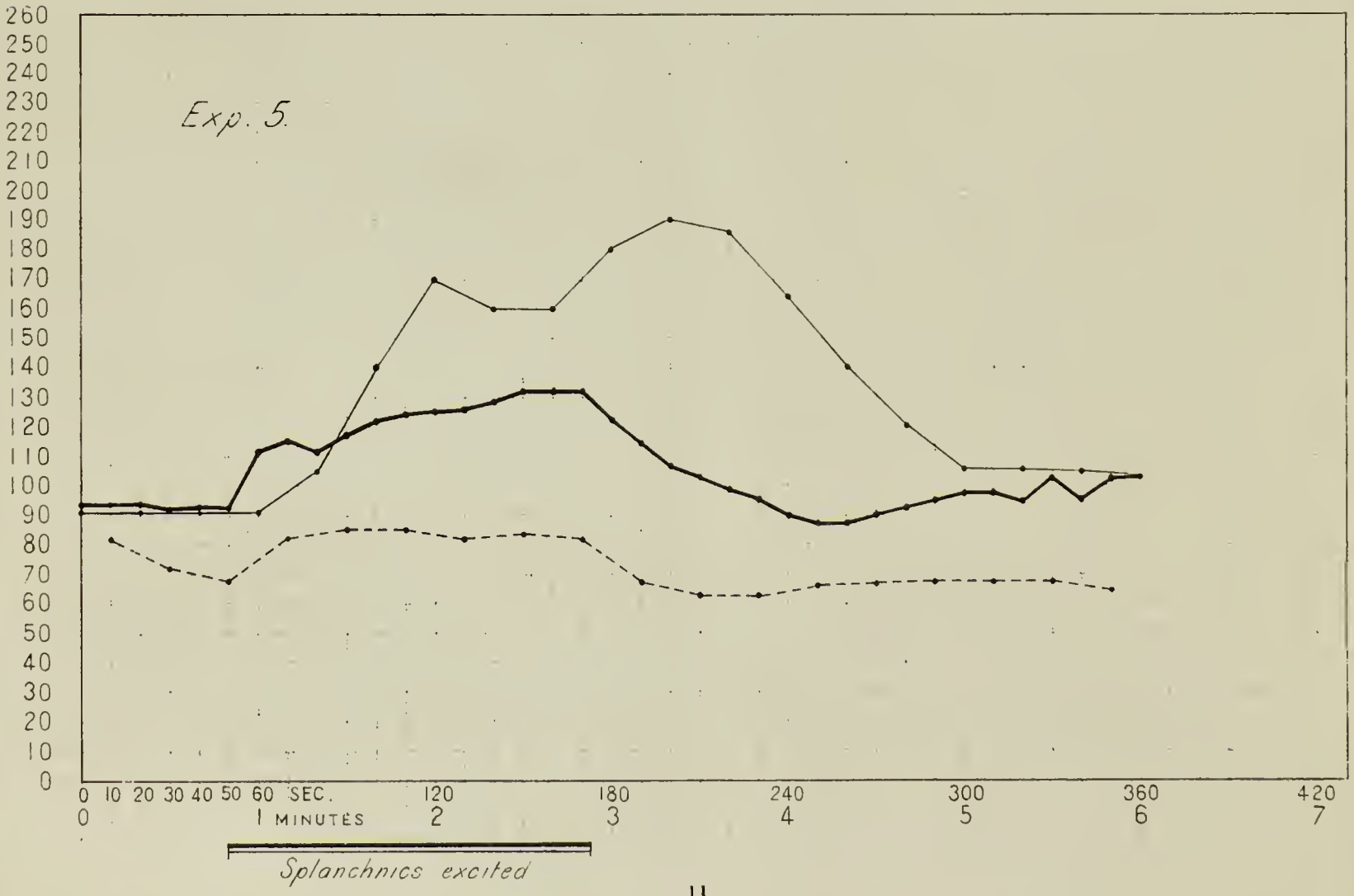
Fem. Art.	Portal Vein	Inf. Cava
62	77	44[1]
60	82	32
55	87	34
Injection begun.		
72	101	36[2]
64	115	46[2]
68	—	—[2]
65	147	90[2]
70	167	124[2]
Injection ended.		
70	157	102
72	153	86

Fem. Art.	Portal Vein	Inf. Cava
72	151	78
72	150	64
74	150	56
72	153	52
68	187	84[3]
76	197	84
74	187	74
72	187	74[4]
72	110	50[5]
72	109	50
84	82	44[6]
86		

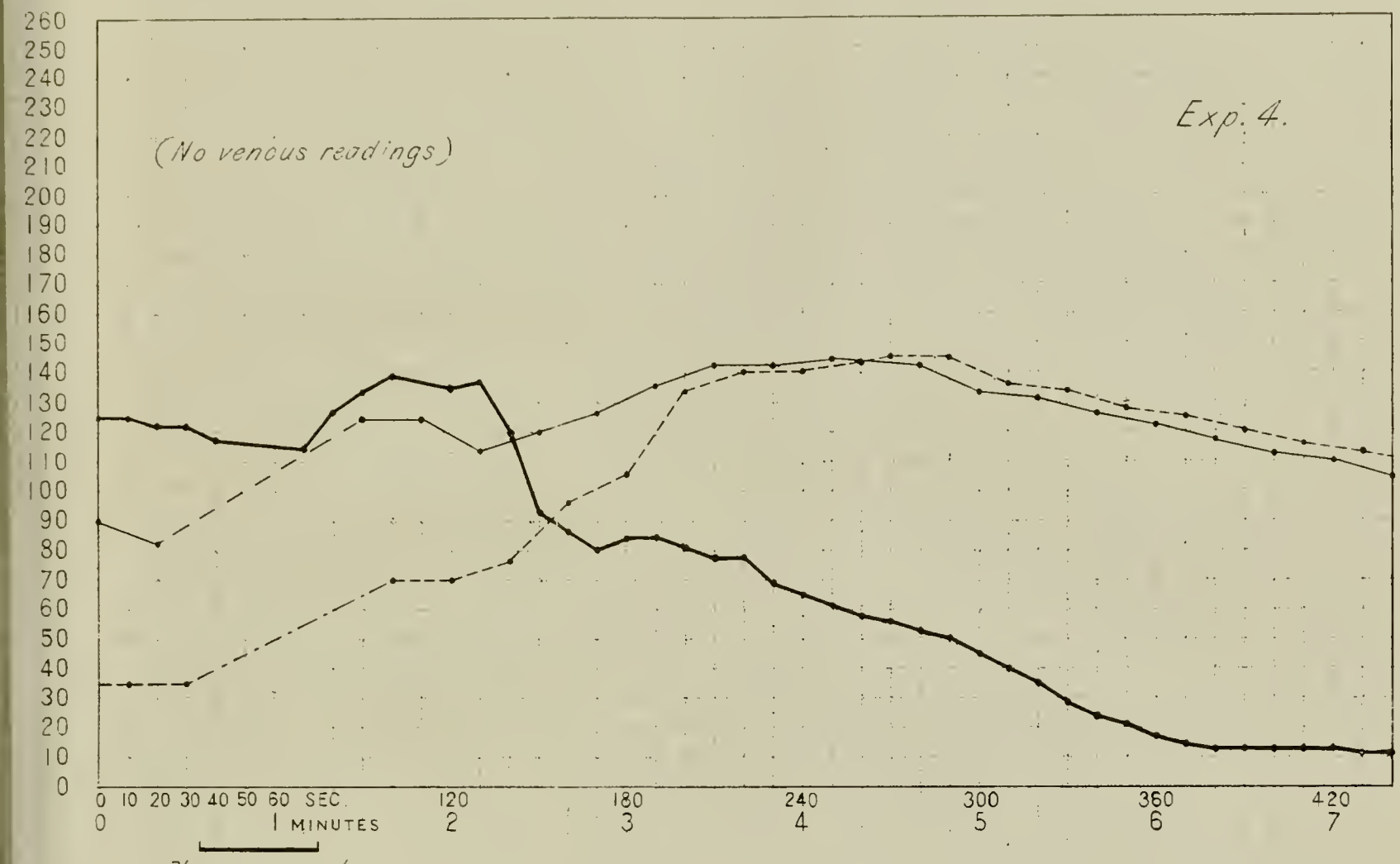
[1] C. At 3 h. 17 m. [2] Injection of 350 c.cm. normal saline (·75 %).
[3] After deep inspiration. [4] At 3 h. 26 m. [5] At 3 h. 36 m. [6] At 4 h. 6 m.


Exp. I.
Heart not beating, Pinched rhythmically
No venous readings
A pause of two minutes to recover circulation.
Heart beating spontaneously & regularly.
260 250 240 230 220 210 200 190 180 170 160 150 140 130 120 110 100 90 80 70 60 50 40 30 20 10 0
0 10 20 30 40 50 60 SEC.
0 1 MINUTES 2 3 4 5 6 7
120 180 240 300 360 420
Vagus excited

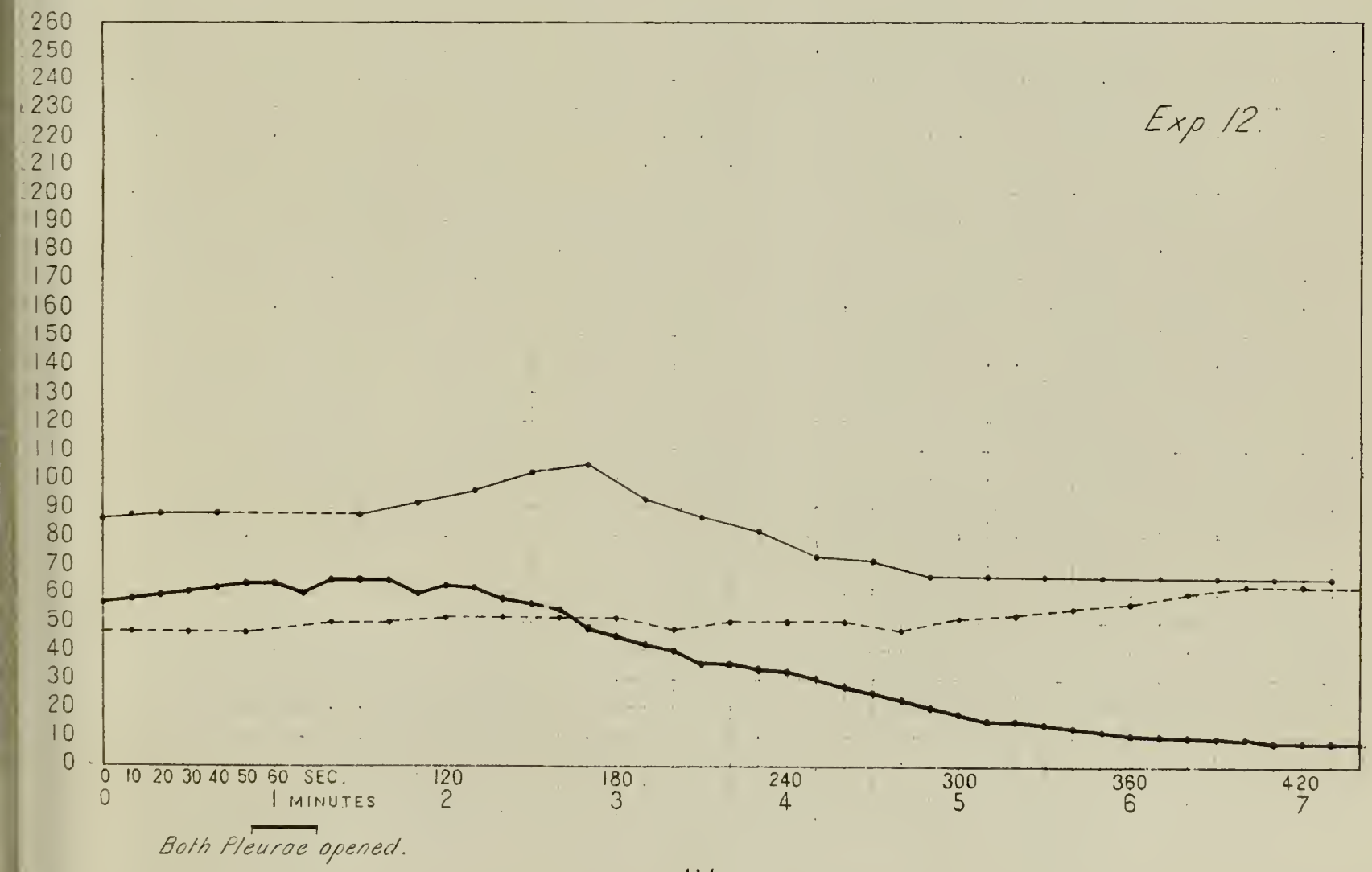
I


Exp. 5.
260 250 240 230 220 210 200 190 180 170 160 150 140 130 120 110 100 90 80 70 60 50 40 30 20 10 0
0 10 20 30 40 50 60 SEC.
0 1 MINUTES 2 3 4 5 6 7
120 180 240 300 360 420
Splanchnics excited

II



III



IV

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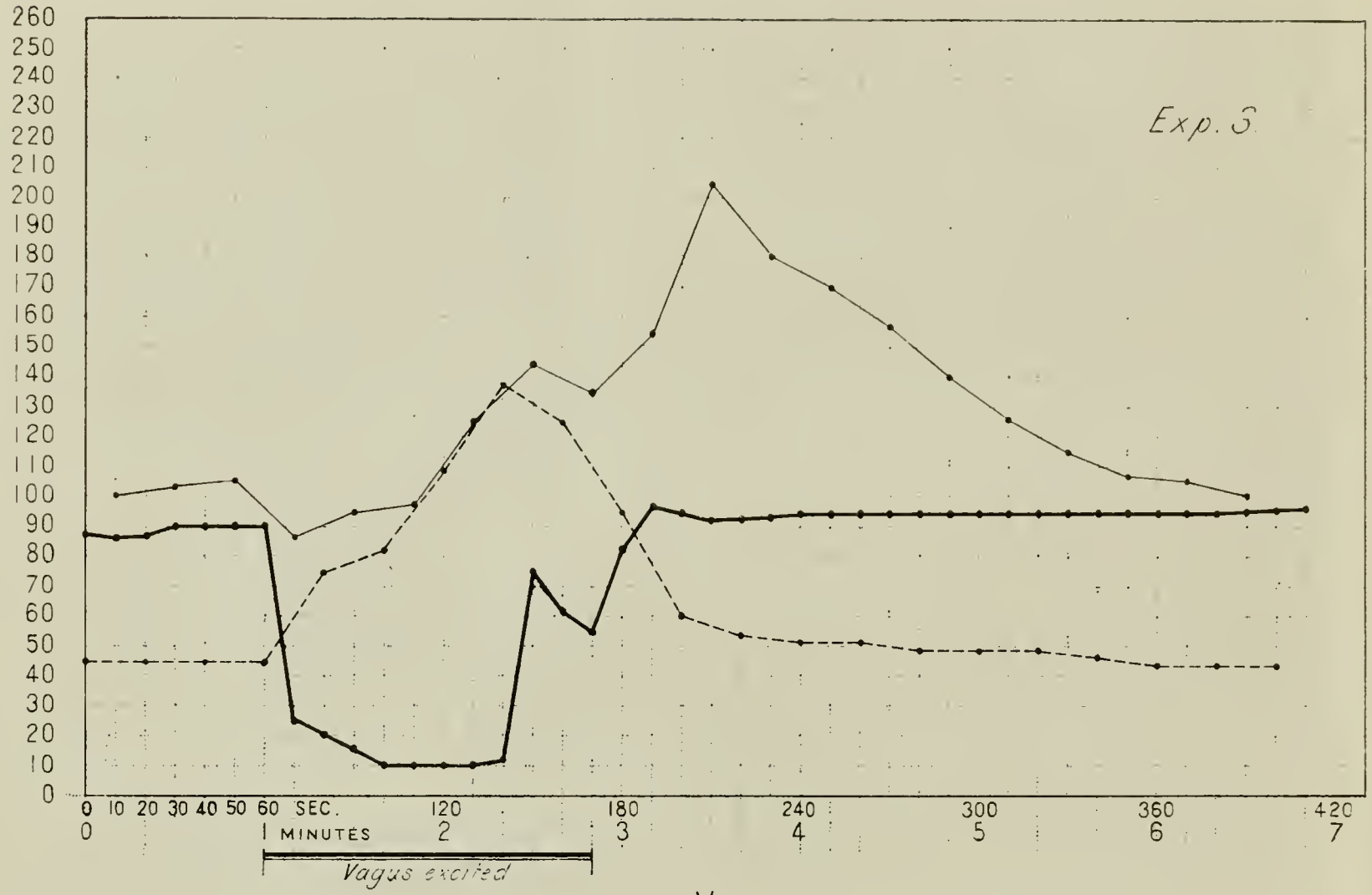
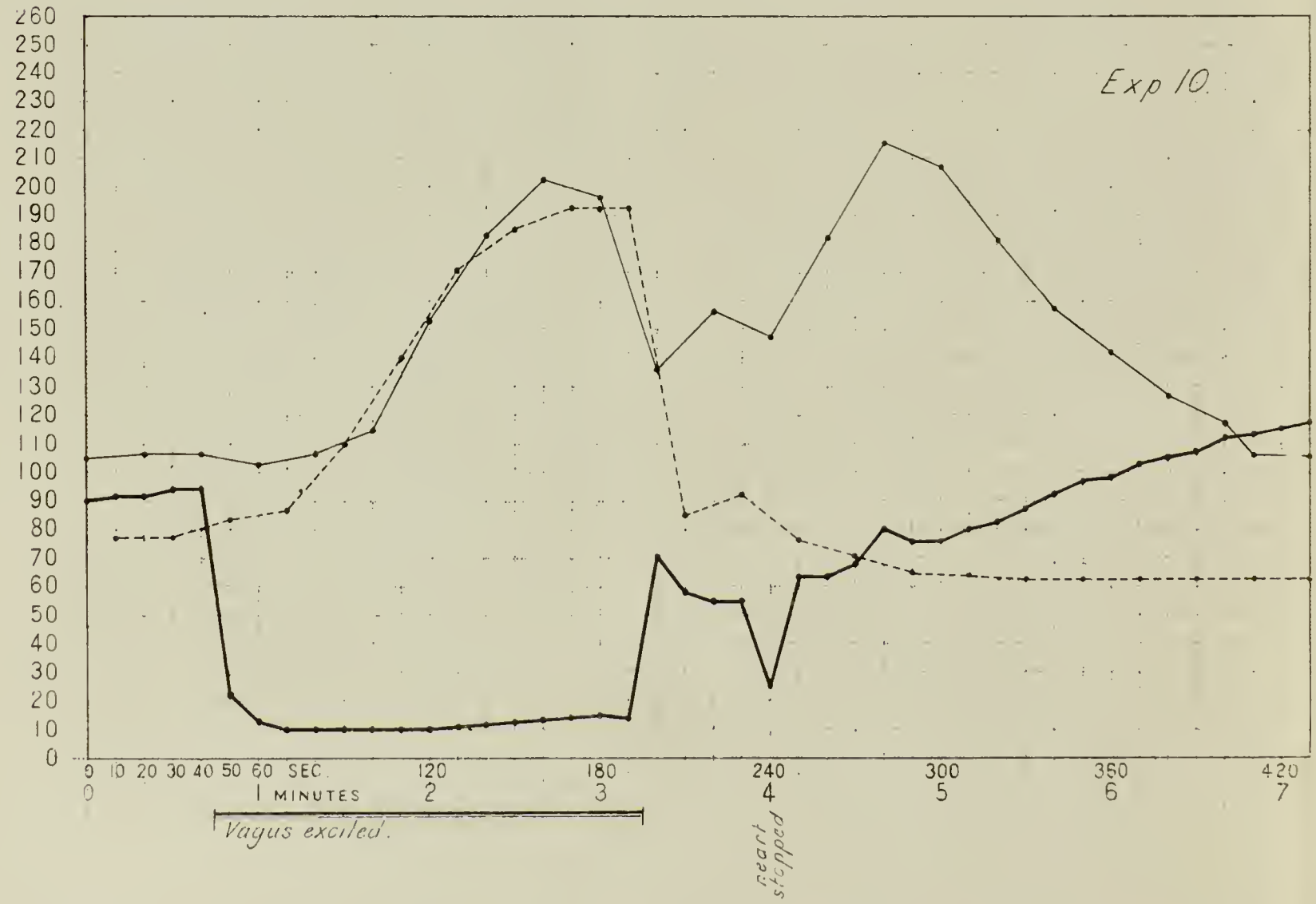
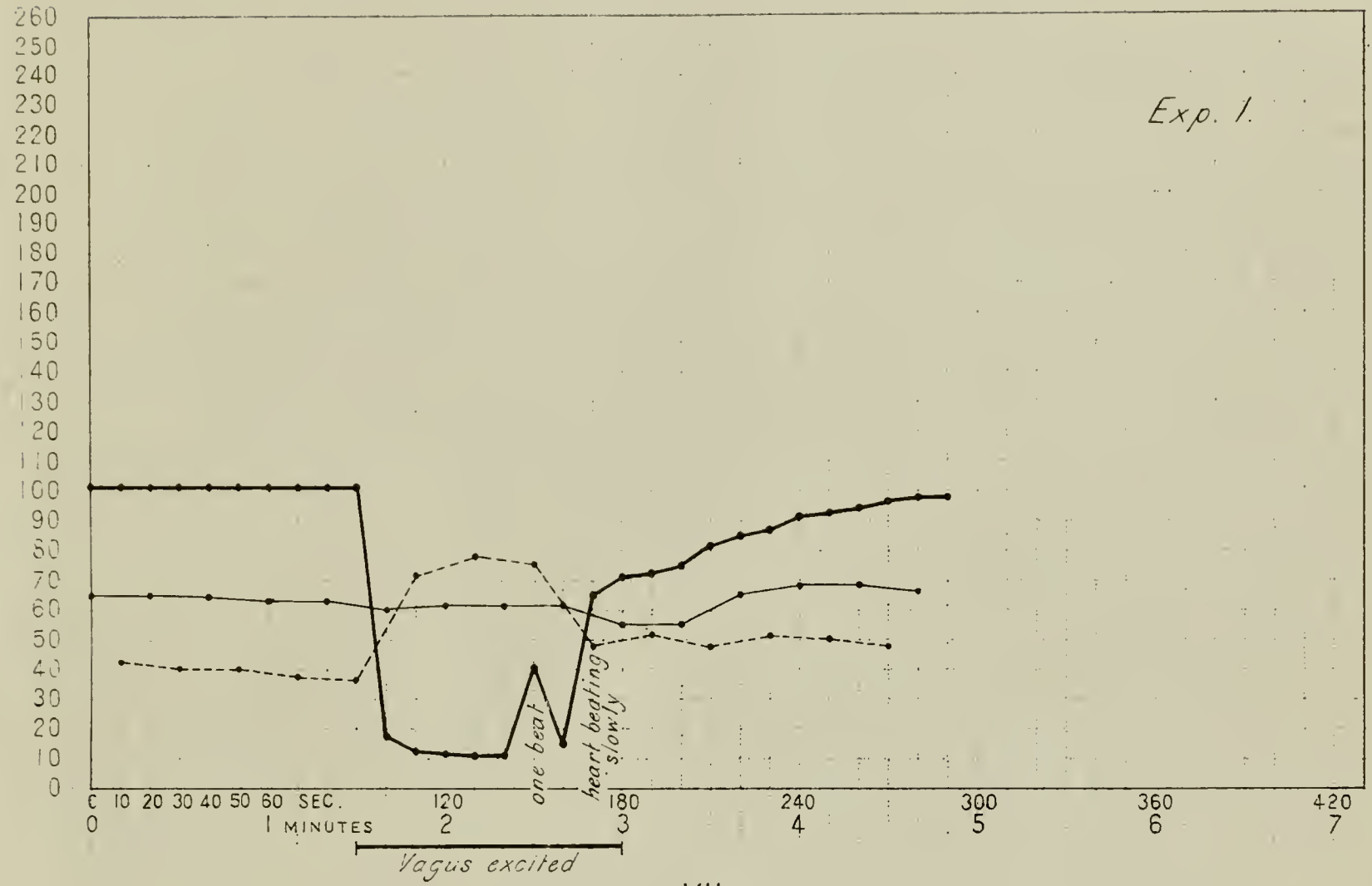

Exp. 3.
260
250
240
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
0 10 20 30 40 50 60 SEC. 120 180 240 300 360 420
0 1 MINUTES 2 3 4 5 6 7
Vagus excited

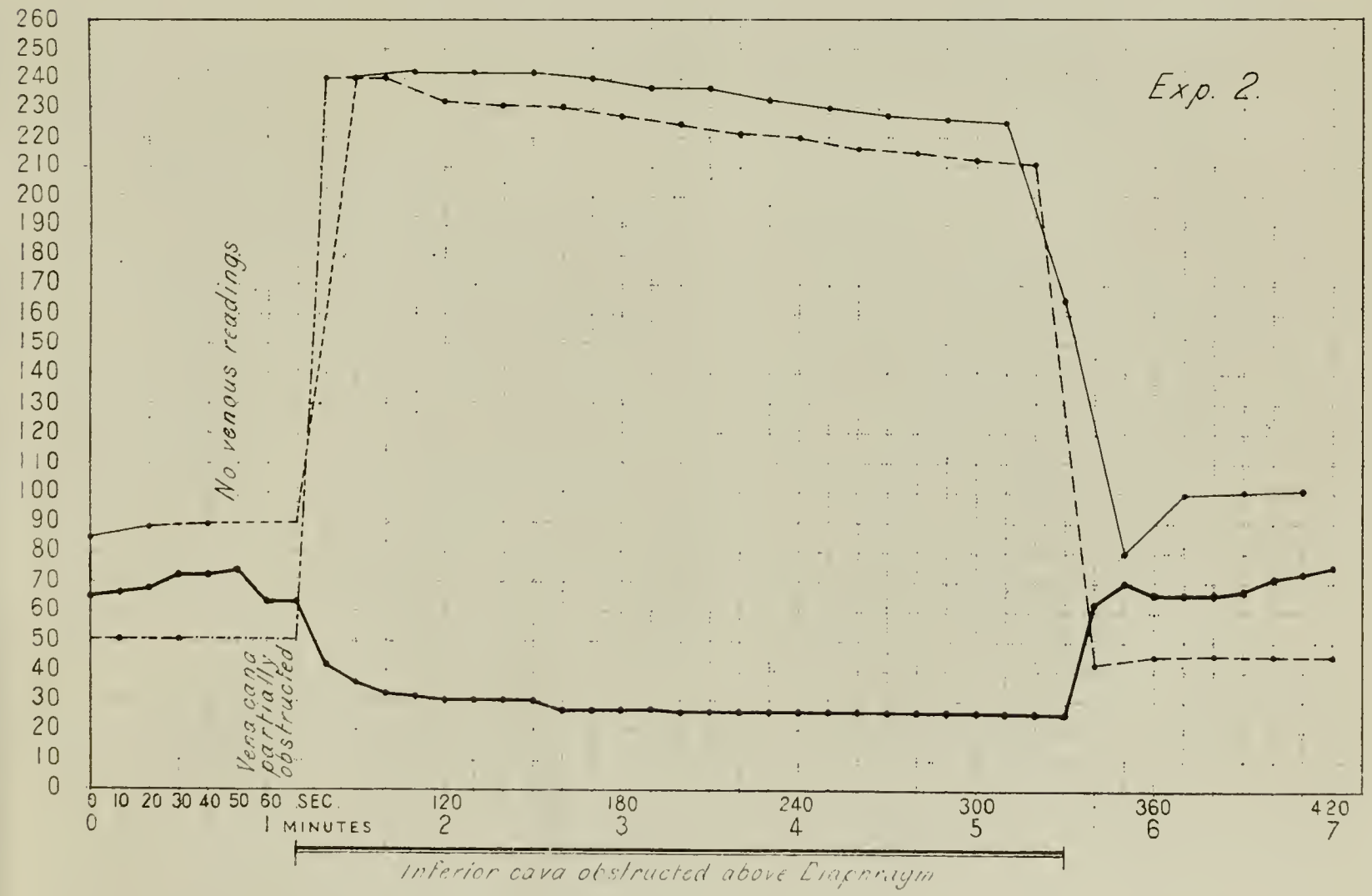
V


Exp 10.
260
250
240
230
220
210
200
190
180
170
160.
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
0 10 20 30 40 50 60 SEC. 120 180 240 300 360 420
0 1 MINUTES 2 3 4 5 6 7
Vagus excited.
Heart stopped

VI



VII



VIII

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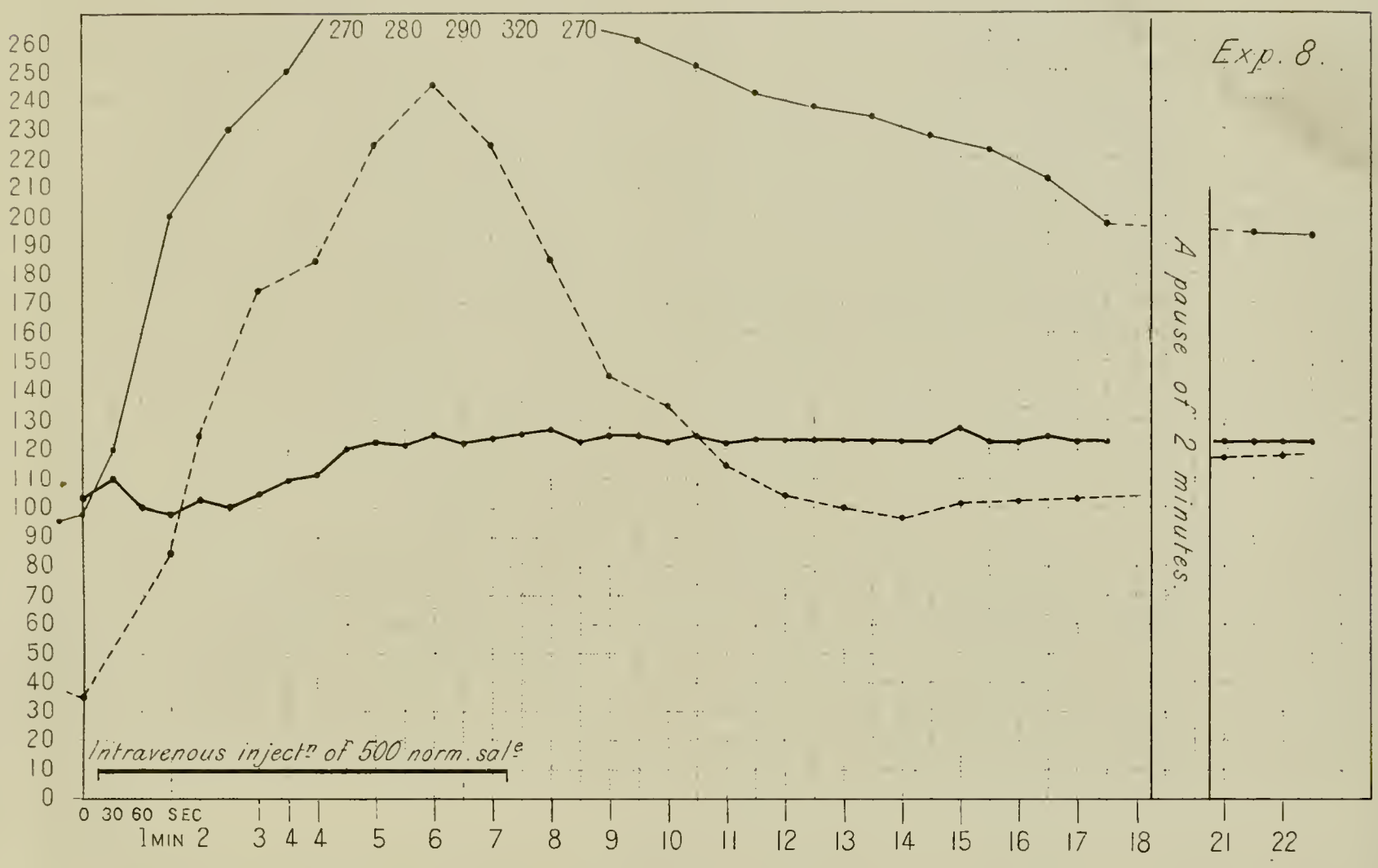
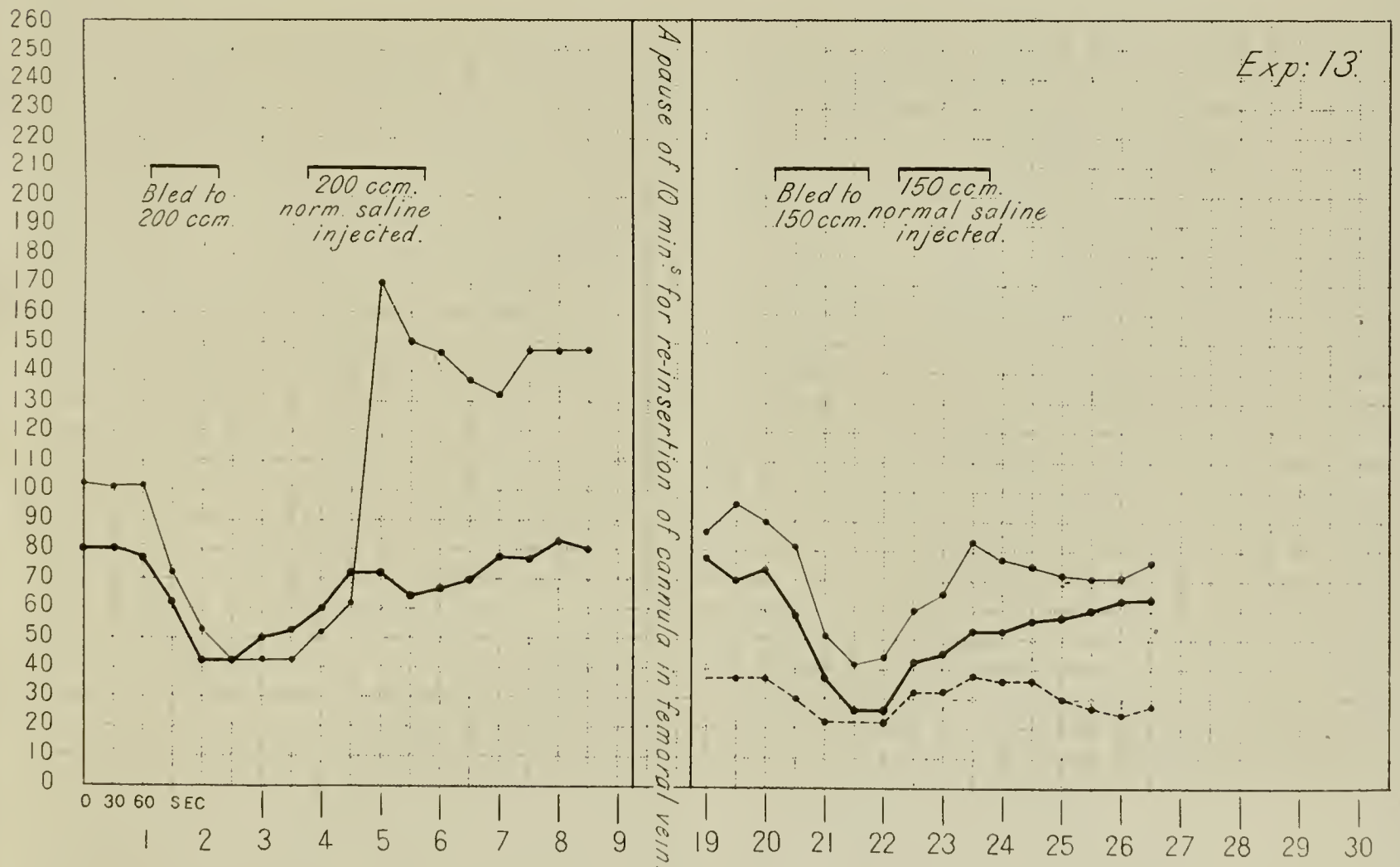


IX



X

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